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A Journal of Neurology

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THE ADVANCEMENT OF LEARNING

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THE ADVANCEMENT OF LEARNING

BRAIN.

PART I., 1909.

Original Articles and Clinical Cases.

ON THE EXACT ORIGIN OF THE PYRAMIDAL TRACTS IN MAN AND OTHER MAMMALS.

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LITTLE or no precise information can be obtained on the exact origin of the pyramidal tracts in even the later editions of the most authoritative text-books on the anatomy of the central nervous system.

According to Edinger [10] "die Tractus cortico-spinales entstammen der Rinde der Zentralwindungen und des Paracentrallappens"; and Obersteiner's [23] account of their origin is as indefinite. According to Dejerine [8] they come from the upper five-sixths of the Rolandic convolutions, the neighbouring portions of the frontal and parietal gyri, and from the paracentral lobule. Von Monakow [20] writes: "Soweit meine eigenen mikroskopischen Erfahrungen reichen scheint die hintere Zentralwindung sich an dem Ursprung der Pyramidenbahn zu beteiligen, ebenso wie auch Abschnitte des Gyrus praecentralis," but "der Löwenantheil des Ursprungsgebietes der Pyramidenbahn in die vordere Zentralwindung, sowie in den Lobus paracentralis fällt."

In van Gehuchten's book [11] it is stated that the cells of origin of the pyramidal tracts occupy in the cerebral cortex "une zone nettement déterminée, appelée *zone motrice de Charcot.* Celle-ci est située autour de la fissure de Rolando et correspond à l'écorce grise de la circonvolution centrale antérieure, de la circonvolution centrale postérieure, du lobe paracentral, et de la partie voisine des circonvolutions frontales." Finally, Hoche [15] may be cited: "Das Rindengebiet von dem aus secundäre Degeneration der eigentlichen Pyramidenbahn erzeugt werden kann, umfasst die oberen zwei Drittel der Centralwindungen und das Paracentrallappchen; Fasern aus dem unteren Drittel, von der Insel, und aus den Centralwindungen unmittelbar anstossenden Teilen der Hirnswindungen stehen der Pyramidenbahn in Bezug auf Verlauf und Function sehr nahe."

From these extracts, and many others which might be quoted, it is evident that not only is the cortical origin of the pyramidal fibres not accurately known, but that the descriptions given of it are vague and discordant. It may be pointed out that all the above-quoted authors include the posterior central gyrus partly or wholly within the area of origin of the pyramidal fibres, and that none of them state from what cells of the cerebral cortex these cortico-spinal fibres actually arise.

Other authors have assumed that the origin of the pyramidal tracts is co-extensive with the so-called "excitable motor cortex"—the limits of which are still a matter of question—or with an area of cortex distinguished by certain structural peculiarities. And several, as Bolton [4] and Campbell [7], have gone further and assumed that the cortico-spinal fibres spring from certain characteristic cells which in distribution are limited to part of the "motor cortex"; but evidently the fact that certain cells are found only in a part of the excitable physiological area is, in itself, no proof that the chief efferent motor tracts take origin from them. Schäfer [25] makes the statement that the giant Betz cells of the cortex give origin to the pyramidal tracts, and undergo chromatolysis after section of these fibres. Unhappily he does not describe the distribution of these cells, nor give references to the work on which his statement is founded.

Recognizing, then, how little is known of the exact origin of the pyramidal tracts, it seemed to us advisable to institute an investigation into the matter. Our main objects have been to determine:—

I.—From what regional area of the cerebral cortex the cortico-spinal tracts arise.

II.—From what cells of this region they take origin.

III.—Is the area of origin of the cortico-spinal tracts co-extensive with the so-called "excitable motor cortex"?

IV.—Does this area coincide with any "structural cortical field," such as has been mapped out in man and various mammals, especially by Campbell and by Brodmann?

II. METHODS.

The method we have adopted has been to examine the cells of the cerebral cortex at some period after section or destruction of the cortico-spinal tracts. The period allowed to elapse between the division of these fibres and the sacrifice of the animal varied from five to 157 days; in the human cases investigated death resulted 108 and 229 days respectively after the accidents which divided the pyramidal tracts in the upper region of the spinal cord.

To insure accurate results it was evidently essential to use cases in which the pyramidal tracts were the only cortico-fugal fibres which were affected. In our experimental material, therefore, except in one lemur, the cortico-spinal tract of one side was divided by hemisection of the spinal cord in the higher cervical segments, and for investigation of the human brain we have employed only cases in which these tracts were interrupted by an acute transverse lesion of the spinal cord. Our human material has been restricted by the fact that to justify the conclusions we wish to draw it was necessary, in the first place, to use only cases in which there was a lesion completely interrupting all the pyramidal fibres; and, in the second, only cases in which the lesion was acute. The latter condition appeared necessary, as even in the peripheral neurones, in which they have been most carefully studied, the reactionary cell-changes on which we have relied in this work occur in their most typical form after acute axonal lesions, and they may not appear at all, or only in modified form, when the interruption of the fibres has resulted from a slowly progressive lesion. In a later communication, however, we hope to show that the cortical changes which follow such spinal lesions, and those which are associated with many forms of system-degeneration of the upper motor neurones, entirely confirm the conclusions we shall state in the present paper.

Immediately after the sacrifice of each animal the brain was carefully removed, and the medial and lateral surfaces of both its hemispheres were photographed in natural size, or traced carefully and drawings made of them. In several cases the anæsthetized animal was transfused with physiological saline solution and immediately injected with one of the well-known fixatives before removal of the brain and its division into blocks. Thus the nerve-cells were actually fixed during life; the importance of this with regard to the subsequent examination of the minute structure of these cells can be scarcely sufficiently emphasized. In the remaining cases, after a preliminary hardening of the whole hemispheres, the Rolandic regions, including the anterior and posterior central convolutions and the neighbouring portions of the frontal and parietal lobes, were cut out and divided into suitable blocks, the position of each block being accurately marked on the corresponding photographs or drawings. In the carnivora relatively much larger areas of cortex, including the whole of the cruciate region, were treated in the same manner. Each of the blocks thus prepared was cut more or less completely into serial sections in either paraffin or colloidin, and each section was stained by some modification of Nissl's method.

In our experimental material the sections of the affected cortex—that is, that of the hemisphere of the opposite side to the spinal lesion—were carefully compared with sections from the corresponding parts of the opposite hemisphere; but as in our human cases both pyramidal tracts were destroyed, and therefore the cortex of each hemisphere equally affected, our sections could be compared only with those from similar regions of normal brains.

III.—MATERIAL.

The material on which our investigations have been carried out consisted of representatives of the carnivora—a dog and three cats—two lemurs, two monkeys of the macaque family, a chimpanzee, and two human brains from cases in which there had been severe traumatic lesions of the spinal cord. For comparison with the latter we have also examined the cortex of two cases with almost complete destruction of the internal capsule and of part of the basal ganglia by lesions of vascular origin.

The nature and the characters of the histological changes we found, in one or other of the stages, in the cells of the cerebral cortex of all these animals may be briefly described here. They consisted essentially of that process which is generally known as retrograde chromatolysis or *reaction à distance*, but its salient features, as it was met with in these cells, deserves some notice.

The cells in which we found these changes and their cortical distribution will be fully described later; here it is only necessary to mention that the characteristic alterations were limited to the giant-cells which distinguish the infra-granular layer of large pyramidal cells of a small region of the cortex—in fact, to those which were originally described by Betz, and are now frequently known by his name.

After complete sections of one pyramidal tract in the higher cervical segments of the cord the changes in these cells appear relatively early. In a cat five days after this operation they were distinct though slight; in a few of the largest cells from the posterior wall of the cruciate sulcus of the opposite hemisphere there was slight disintegration of the tigroid clumps near the axonal origin and in the perinuclear zone. In a dog twenty-three days after the operation many of these cells were in advanced chromatolysis; their cell-bodies were more rounded than normal, the tigroid clumps were more or less disintegrated into fine granules, especially in the perinuclear and axonal regions, though the dendrites still contained many well-formed chromatophilic masses; their nuclei were generally eccentric and often irregular in shape, and a certain proportion of the cells were distinctly swollen, but the swelling of the cell-bodies was not a constant or very prominent feature. At this stage there was already some shrinkage or atrophy of some of the affected cells, and their dendrites began to disappear and break off. Cells in the more advanced stages of degeneration were frequently surrounded by several neurophages. Finally, even twenty-three days after the operation a considerable number of the affected cells had already disappeared, or only indistinct remnants of them were visible. In a cat killed twenty-nine days after hemisection of the left side of the cord changes similar, but somewhat further advanced, could be seen in the right cerebral hemisphere; here swollen cells were less numerous, the majority being shrunken and atrophic, and the proportional disappearance was much higher.

In a macaque monkey killed forty-nine days after spinal hemisection the cortical changes were very similar to the above, but in another animal which was allowed to survive the operation 157 days the most prominent change was a great reduction in the number of giant-cells in the opposite hemisphere—in fact, only about one-thirteenth of the normal number could be seen; a few chromatolytic and degenerated forms were still visible.

In our human cases the cell-changes were more or less parallel to those we observed in our experimental material, but in relation to these a factor must be considered which appears to have an influence on the intensity of the chromatolysis and the rate of its course. This is the relative distance of the axonal lesion from the cell, or rather the relative amount of the axis-cylinder that is cut off from the cell by the lesion. It has been repeatedly observed by various experimenters that the reaction of the cell is more early and more intense the nearer the axis-cylinder is injured to its origin. In both our human cases the lesion lay in the seventh cervical segment of the cord, and consequently destroyed only the terminations of the cortico-spinal fibres which innervate the spinal centres for the wrist and fingers, but cut off relatively large portions of the neurone units which pass from the upper portion of the Rolandic region to the lumbo-sacral cord. This may be the explanation of the fact that in these brains the cortical cell-reactions were appreciably less intense and appeared later in the regions which we know send fibres to the cervical enlargement of the cord, than in those connected with the lumbar and sacral segments.

Although chromatolysis was the most characteristic change, it seems that a certain number of the affected cells merely atrophy and eventually disappear, some of the tigroid bodies persisting almost to the end. A smaller proportion, variable in different animals, undergo a pyknomorphic form of degeneration; these shrink but stain very intensely and almost uniformly, and the nucleus may be distorted, but is rarely, especially in the early stages, much displaced from its normal position near the centre of the cell.

Carnivora.

We have made use in this research of the nervous system of one dog and three cats.

Dog.—The animal was killed twenty-three days after section of the left side of the spinal cord in the first cervical segment. On microscopical examination it was found that the whole of the lateral column, the greater part of the dorsal column, and part of the ventral column had been completely divided, while there was almost complete destruction of the rest of the left side of the cord by softening. The left pyramidal tract, as shown by the Marchi method of staining, appeared totally degenerated below the level of the lesion.

The cerebral cortex of the anterior half of each hemisphere was, after being divided into suitable blocks, cut into serial sections.

On the mesial surface of the right hemisphere the cell-changes were limited to a triangular area with its base, which measured 8 mm., on the apical margin of the hemisphere and its anterior edge along the bottom of the sulcus cruciatus almost as far, in this particular hemisphere, as its junction with the sulcus splenialis (fig. 1). The changes were entirely limited to the giant-cells of the infra-granular layer. These were, in the first place, much less numerous than in the corresponding area of the opposite hemisphere; to determine the actual loss of these cells they were counted in a complete series of sections in both hemispheres. In the right—that is, in the affected side—all cells that could be recognized by a relatively low magnification were included, though, as will be shown, a considerable proportion of these were in some stage of chromatolysis. This enumeration revealed the fact that, even in the relatively short period that this animal survived the operation (twenty-three days), these cells were reduced to about a fourth or even less of their normal number. In a series of sections taken from near the upper margin of the left hemisphere, and parallel to it, the average number of giant-cells per section was 18·2, while in the exactly corresponding region of the other side

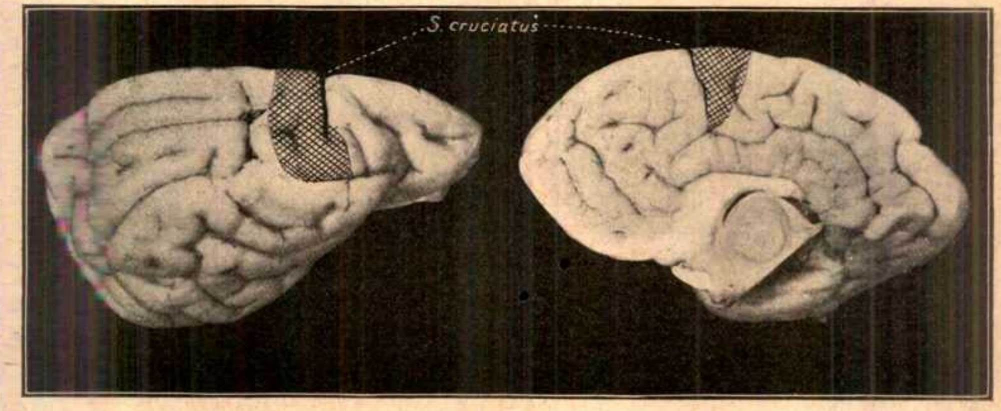

FIG. 1.

Photographs of the brain of the dog; the cross-hatched areas represent the origin of the cortico-spinal fibres.

there were on an average only 3·5 in sections of the same thickness. Further, of these very few could pass as normal; almost all were in some stage of chromatolysis, but a few seemed small or shrunken with their nuclei distorted or misshapen. By examination with higher magnification the remnants of a considerable proportion of the more severely affected cells could be seen, generally only as faint shadows containing no chromatophilic material, and with only distorted and shrunken nuclei. Collections of neurophages were usually present around many of the cells in the more advanced stages of chromatolysis, but no inflammatory or vascular changes could be seen.

Repeated examination failed to discover such changes in any cells save the giant-cells of the infra-granular layer, which correspond to the Betz cells of the human cortex; no numerical loss of, or chromatolytic changes in, cells of any other variety could be detected. Owing to their size alone the affected cells cannot be mistaken, for the supra-granular layer contains no cells which are even relatively large, and in size, shape and structure the cells with which we are here concerned are characteristically different from the few large pyramidal cells of the layer in which they lie.

The superficial extent of the changes on the lateral surface of the hemisphere is best understood from fig. 1. It includes, approximately, the anterior two-thirds of the gyrus sigmoideus posterior; it does not extend anterior to the bottom of the sulcus cruciatus in the mesial half of this fissure, but further lateralwards it invades its anterior wall and appears on the gyrus sigmoideus anterior about 2 mm. from the lateral extremity of the sulcus. Beyond this the whole area, remaining about the same breadth, slopes somewhat forward and lateralward to the sulcus coronalis, beyond which no definite changes could be discovered. The amount of the anterior sigmoid gyrus which is affected is thus small. A considerable part of the affected area lies in the walls of the sulcus cruciatus. The changes which characterize this area were identical with those which have been described in the contiguous part of the mesial surface of the hemisphere, and the proportion of the cells affected was approximately the same. The changes were most pronounced in the posterior wall of the cruciate sulcus and in the anterior lip of the posterior sigmoid gyrus, in the regions in which the giant-cells are normally most numerous. Lateral to the extremity of the sulcus cruciatus the proportion of these cells which are unaffected appears to be somewhat larger.

The area we describe does not extend as far forwards as the small precruciate sulcus, and in the intervening area, as well as in front of this fissure, large intact cells, similar in appearance to those which are affected, were present in considerable numbers. In this region—that is in the lower part of the gyrus sigmoideus anterior—the frontal limit of the affected area is not sharp or definite. Its posterior border, on the other hand, is fairly sharply defined; it coincides practically with the distribution of the giant-cells, though in the most lateral part of the posterior sigmoid gyrus, in the neighbourhood of the ansate sulcus, a small number of large cells remain unaltered.

No pathological changes were found in any other region of the cortex.

Cat 2.—Of the three cats which were operated on the most suitable for the detailed description of the resulting cortical changes was an animal which was killed twenty-nine days after section of the left side of the cord in the first cervical segment. The extent of the operative lesion was determined in serial sections; the whole of the left dorsal and lateral columns, and the greater part of the ventral column, were completely divided, and the only portion of the latter that escaped the cautery was softened. The right side of the cord, excepting a small strip of the dorsal column along the septum, was uninjured. The degeneration of the left pyramidal tract in the cord below the lesion was complete.

On the mesial surface of the right hemisphere the affected cortex occupied a triangular area between the margin of the hemisphere and the sulcus cruciatus, as well as the posterior wall of the latter. It extended about 4 or 5 mm. along the fissure—that is somewhat more than half-way to the level of the anterior extremity of the sulcus splenialis (fig. 2). The cell-changes in this area were of exactly the same nature as, but somewhat further advanced than, those in the dog. Here, also, only the large cells which characterize this region of the cortex were affected. These are, as compared with all the other cells of the cortex, enormous in the cat; many measure 30 by 50 μ, and even more. All these giant-cells were counted in serial sections in the left hemisphere, and thus the normal average number per section for each region was determined. The average number of normal cells, those showing any degree of chromatolysis being excluded, per section of the corresponding areas of the right hemisphere was similarly determined, and on comparison it was found that only from a fifteenth to a twentieth of the giant-cells of the right side were unaffected. In the anterior third of this area the average number of normal giant-cells per section was

12·6 in the left hemisphere, only 0·8 on the right; 10 and ·55 respectively in the middle thirds, and 7 and ·4 in the posterior thirds. The affected cells were in various stages of chromatolysis, and often only shadows or remnants were visible; the latter were often surrounded by clumps of neurophages, and small collections of these were occasionally the only indications of the positions from which chromatolytic cells had already disappeared.

The distribution of the pathological changes on the convexity of the hemisphere is best seen in fig. 2. At the apical margin its area lies entirely behind the sulcus cruciatus, but further lateralwards it crosses the bottom of this sulcus and appears on the anterior sigmoid gyrus, about 3 mm. from the mesial margin. From here its frontal margin advances lateralwards and frontalwards almost to the sulcus orbitalis.

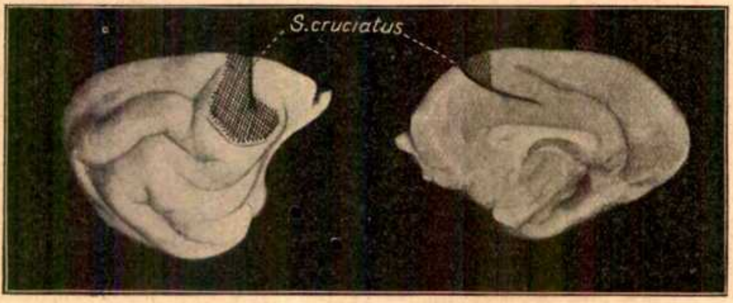

FIG. 2.

The brain of a cat; the shaded portions represent the areas of origin of the cortico-spinal fibres.

The altered cells extend backwards almost, but not quite, to the sulcus ansatus, and lateralwards almost to the sulcus coronalis. This lateral margin, and especially its frontal part, was difficult to define, as here large numbers of normal cells appeared intermixed with others in chromatolysis. No definite changes could be recognized lateral to the sulcus coronalis.

The changes which characterized the area we have just defined were identical in nature and practically in degree with those discovered on the mesial surface. Between the margin of the hemisphere and the lateral extremity of the sulcus cruciatus only from 30 to 40 per cent. of the normal number of giant-cells were recognizable by a low magnification, and only about 6 per cent. or 7 per cent. of those present were unaffected, the remaining being in various stages of chromatolysis.

Beyond the lateral extremity of the sulcus cruciatus the proportion of normal cells rose considerably, and towards the lip of the sulcus coronalis it reached about 25 per cent.

Repeated careful examinations of our sections has confirmed our first observations—that it was only the giant-cells of this area which were unaffected; no numerical loss could be detected in the other layers of the cortex, and none of their cells were seen in chromatolysis.

Prosimia.

Two lemurs (*L. catta*) were operated on. In the one the cord was hemisected in the first cervical segment; in the other the left crus was divided. The latter animal proved especially valuable for the purpose of our present research. Immediately after the operation in this case the paralysis of the limbs and trunk of the side opposite to the lesion was almost total, and return of movement occurred much more slowly than in any of our other animals; slight and steady improvement, however, continued till the animal was sacrificed twenty-five days later.

Before death the cortex of each hemisphere was experimentally investigated by electrical stimulation, using both the bipolar and unipolar methods. Though muscular responses were readily obtained by excitation of the appropriate area of the right cerebral cortex (as previously delimited by one of us (W. P. M.) in several other lemurs), no movements whatever could be obtained even by unusually strong currents from any part of the hemisphere of the same side as the divided crus. At the autopsy it was found that the left crus had been completely divided. Systematic histological examination of the cortex of each side was carried out exactly as in our other animals.

Well-marked and characteristic changes were found in the cortex of the cerebral hemisphere on the same side as the divided crus; in fact, scarcely a single normal giant-cell could be found, almost all being in a well-marked or advanced stage of chromatolysis. Moreover, the number of these giant-cells was reduced to about the proportion of seven to sixteen, as compared with the healthy side. In spite, however, of this well-marked affection of the giant-cells, it was by no means easy to delimit accurately the area that contained them, for in the lowly differentiated prosimian brain there is a remarkable absence of definite sulci in immediate relation with the motor area. The most important landmark—which we shall speak of as sulcus x[1]—is often merely a superficial depression, and not infrequently asymmetrically placed in the two hemispheres, or even absent on one side. Further, the affected cells become much less numerous towards the confines of the area, and so sparse and so irregularly scattered that it is difficult to draw a line of demarcation that shall include them all without enclosing small portions of unaffected cortex. The area shown in fig. 3 is, however, sufficiently definite, and includes practically all the affected cells. On the mesial surface it does not extend quite to the callosomarginal fissure, and on the lateral surface it lies almost entirely in front of sulcus x, which, however, it just includes. Affected cells are more numerous in the anterior than in the posterior wall of this sulcus, and very few giant-cells in chromatolysis could be found behind its posterior lip. The area definitely crosses the sulcus frontalis, narrowing as it descends, and almost reaches the sylvian fissure, but its lateral limit is, for the reasons given above, not sharply definable.

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[1] Homologue of the upper part of the Fissure of Rolando, according to Elliot Smith; the sulcus centralis of Ziehen.

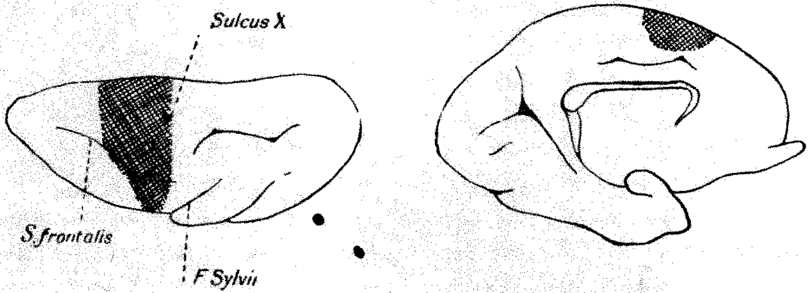

FIG. 3.
The brain of *lemur catta*; the shaded portions represent the areas in which cortical changes were found secondary to section of the left crus cerebri.

In addition to this affection of the giant-cells similar changes were found in a certain number of the ordinary large pyramidal cells of the supra-granular, but more especially of the infra-granular, layers; the affection of these cells was much wider spread than the area which contained giant-cells. The significance of the involvement of these large pyramidal cells will be discussed later.

The Gyrencephalic Monkeys.

Two animals of this class were used for this investigation.

(1) *Cercopithecus callithrix*—The animal was killed 157 days after section by the electro-cautery of the right side of the spinal cord in the first cervical segment. Immediately before it was sacrificed the "motor"

cortex of both hemispheres was investigated under anæsthesia by means of a faradic current, and with the usual precautions. Both the unipolar and bipolar methods were employed. Movements of the left limbs were easily obtained by excitation of the right anterior central gyrus, but to currents of the same strength the posterior central gyrus gave no muscular response. No movements of the limbs could be produced from the corresponding regions of the left hemisphere by a current of this strength, but slight movements of the homo-lateral (left) toes were obtained by the application of a considerably stronger current to the pre-central gyrus above the level of the precentral sulcus, and similar excitation of the gyrus between this level and the bend of the sulcus arcuatus elicited slight movements in the elbow, wrist and fingers of the same side.

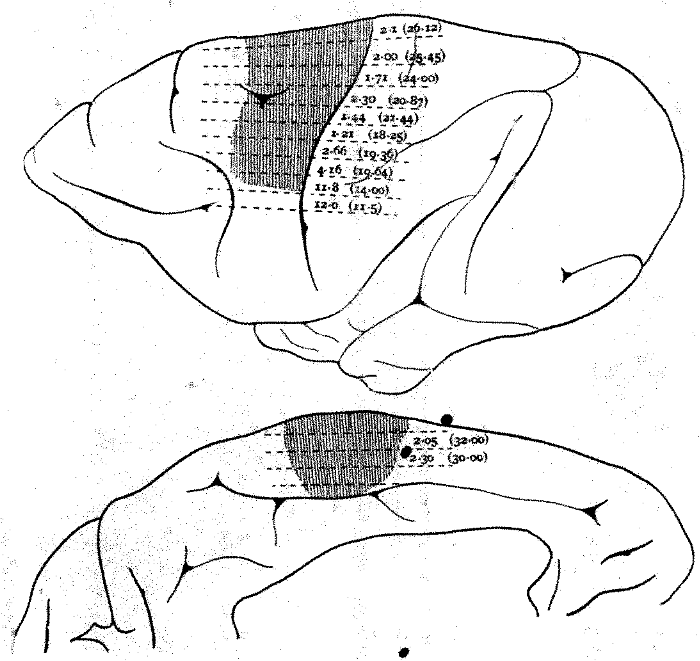

FIG. 4.

Tracings from photographs of the lateral and mesial surfaces of the brain of *cercopithecus callitrix*: the shaded portions indicate the areas in which cortical changes were found after section of the right side of the spinal cord in the first cervical segment. The figures represent the average number of giant-cells per section found at the different levels in this hemisphere; those in brackets the average number per section at the corresponding levels of the opposite hemisphere.

The operative lesion involved practically the whole cross-section of the right side of the cord.

The anterior and posterior central gyri, with the greater part of the frontal lobes and the contiguous portion of the medial surface of both hemispheres, were studied in serial sections stained by thionin. The distribution of the changes which were found in the left hemisphere is represented in fig. 4. On the medial surface it lay between the margin of the hemisphere and the callosomarginal sulcus, extending frontalwards about 13 mm. from the upper end of the sulcus centralis; the area narrowed considerably towards the callosomarginal sulcus, being here scarcely half as broad as at the margin. On the lateral surface it extended to about the level of the genu of the sulcus arcuatus, or to a line drawn from its angle to the extremity of the sulcus intraparietalis. At the apex of the hemisphere its posterior border fell practically along the bottom of the sulcus centralis, but lower it receded slightly from it, ascending the anterior wall of the sulcus. The anterior margin extended from a point on the apex of the hemisphere about 13 mm. in front of the sulcus centralis to the knee of the sulcus arcuatus; the sulcus precentralis superior was included within it.

The most striking change in this area was the enormous reduction in number of the giant-cells of the infra-granular layer. To determine accurately the degree of this change the giant-cells of the right hemisphere were counted in several series of sections, and those persisting in the left were enumerated in the same manner. By comparison of the numbers so obtained from corresponding regions of the two hemispheres it was evident that only from a fifteenth to a twelfth of the normal number persisted in the affected area on the left side. Here and there throughout this area a few remnants of degenerated cells were visible, but they were rare; they generally appeared as shrunken palely staining structures with distorted and often protruding nuclei, and were frequently surrounded by neurophages. Occasionally degenerated cells were represented only by a mass of pigment, and often their site was recognizable only by a group of neurophages, which were generally laden with nerve-cell pigment (fig. 5).

Despite this remarkable affection of the giant-cells we could detect absolutely no change in the ordinary large pyramidal cells of the supra- or infra-granular layers of the cortex of this region, or in the cells of any other layer. There did not appear to be, even on careful comparison with the normal, the slightest reduction in the number of these cells, and no remnants or degenerated forms were visible other than those described above.

The changes appeared to be entirely limited to the area we have

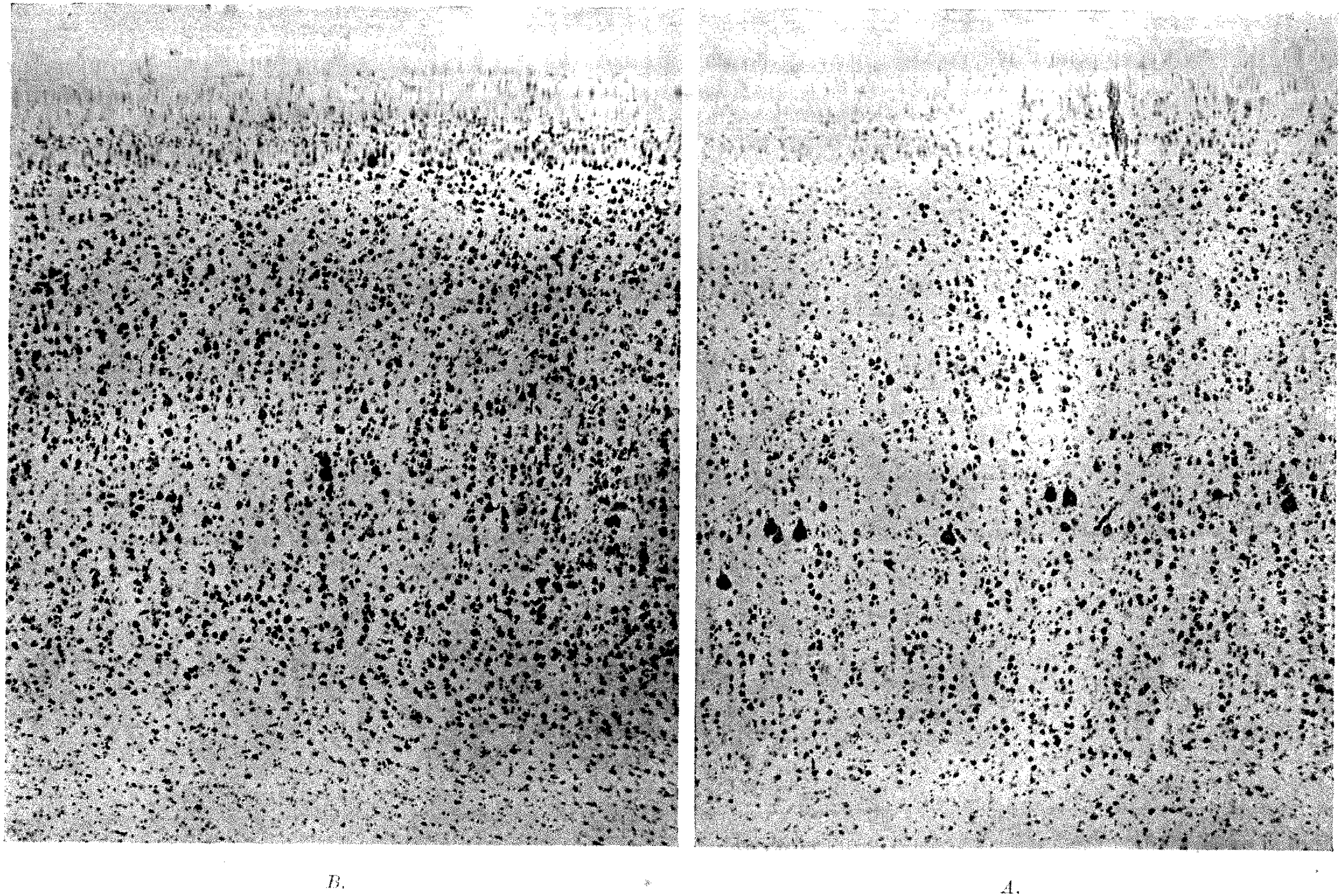

FIG. 5.

A, a microphotograph of the normal precentral cortex (area gigantopyramidalis) of the macaque monkey : B, from a section in a corresponding part of the left hemisphere 157 days after division of the right side of the spinal cord ; only the indistinct remnants of two giant-cells are visible.

defined; certainly no part of the cortex posterior to the sulcus centralis was in any way affected.

(2) *Macacus rhesus.*—The animal was killed forty-nine days after section of the left side of the spinal cord in the first cervical segment. When examined in the microscopical sections it was found that the lesion had almost completely severed the left side of the cord, while of the right side it injured only a small portion of the dorsal columns. The degeneration of the left pyramidal tract was complete below the lesion. The examination of the cortex was undertaken in the usual way, the region of each hemisphere systematically examined including the anterior and posterior central gyri and the greater part of each frontal lobe.

The area in which the characteristic changes were found corresponded closely to—in fact was almost identical with—that mapped out in the previously described animal (fig. 6), though its superficial extent on the surface of the hemisphere was scarcely so broad; at the apical margin it reached 11 mm. in front of the upper extremity of the sulcus centralis. It extended downwards along the sulcus centralis almost to the genu of the sulcus arcuatus, not quite to a line connecting this with the extremity of the sulcus intraparietalis. The details of the changes which mark out this area differed considerably from those described in the callithrix monkey, owing to the much shorter period the animal was allowed to survive the operation; they consequently resembled more closely those we have described in the dog and cats which were killed a much shorter time after the operation.

There was, in the first place, a considerable reduction in the number of the giant-cells of the infra-granular layer; on an average only between a third and a half of the normal number persisted, even including in our enumeration several which were in advanced stages of chromatolysis. The actual number of intact cells was small, approximately the same as in the callithrix monkey—that is, only about, or less than, a twelfth of the normal. The affected cells were, as a rule, in advanced chromatolysis, with complete dissolution of the tigroid clumps and excentration of the nucleus, but very few were swollen; in fact, many were shrunken and atrophic. In many places the degenerated remains of cells could be seen surrounded by neurophages.

As in the specimens previously described, it was only the giant-cells of the infra-granular layer of the cortex which were affected; in such a case as this in which chromatolysis was the predominant change it would have been scarcely possible to fail to detect the specific change in any of the other cells if they had been similarly altered. In this specimen, too, there could be no doubt that the characteristic changes did not extend to the cortex on the posterior wall of the sulcus centralis, or to that covering the posterior central gyrus.

Chimpanzee.

Two animals were operated on by one of us (W. P. M.) in the Physiological Laboratory of the University of Liverpool; we wish to record our cordial thanks to Professor Sherrington, who kindly gave his valuable help at these operations. In each the lateral pyramidal tract was completely divided in the third cervical segment of the cord, but, unfortunately, though both operations were successful, one of the animals died on the third day from influenza. The second animal made a complete recovery, and was allowed to survive exactly four weeks.

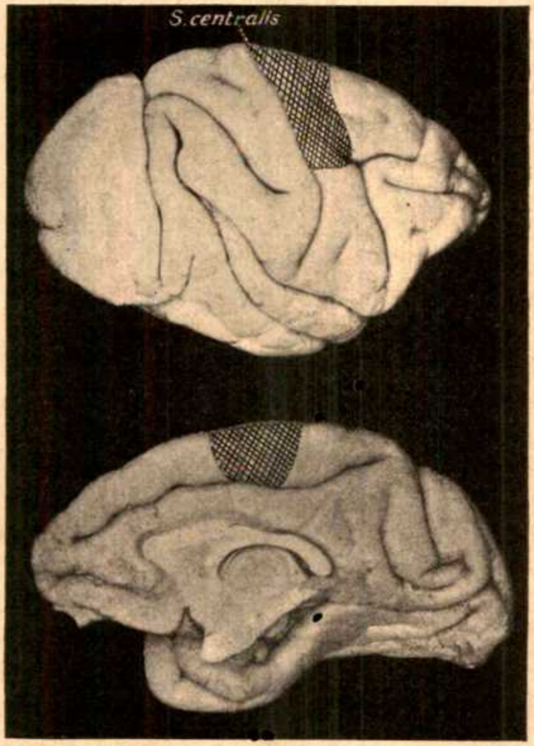

FIG. 6.

The brain of a macaque monkey, the shaded areas representing the regions in which cortical changes were found after division of the left side of the spinal cord in the first cervical segment.

Immediately after the operation there was total paralysis of the arm and leg on the side of the lesion, and serious interference with the movements of the same side of the trunk. But in about thirty-six hours a visible return of power began to set in and steady improvement continued for four weeks, when to a layman the animal would have seemed in possession of all ordinary power of movement; walking was apparently normal, and the affected forelimb was used in climbing and not infrequently in taking food. The vasomotor and sensory phenomena which resulted from the lesion need not be considered here.

Twenty-eight days after the operation the animal was again placed under chloroform, and the cortex of both cerebral hemispheres was investigated by electrical stimulation during some five or six hours. Both the unipolar and bipolar methods of stimulation were employed, and by a simple arrangement it was made possible to employ the same battery and secondary coil for either method at any moment, so that exactly the same strength of current could be applied as desired through either the unipolar or bipolar electrode.

Stimulation of the motor cortex of the hemisphere of the same side as the spinal lesion gave results which agreed closely with the classical researches of Grünbaum and Sherrington [13] on the anthropoid apes. Further details of this particular investigation will be given in another place, but here we may say that no response whatever was obtained from any part of the posterior central gyrus, and that the area which responded to stimulation by a minimal current was limited to the anterior central and the paracentral gyri, and to the immediately adjoining portions of the superior and middle frontal convolutions. From the cortex of the other cerebral hemisphere—that contra-lateral to the spinal lesion—no movements of the limbs of either side, or of the trunk, could be obtained during the first three hours of stimulation, but after that time, when the cortex was presumably passing into a state of greater excitability, slight movements of the contra-lateral toes, knee and hip were obtained, and also, but to an even less degree, of the shoulder and fingers.

Homo-lateral movements could not be elicited from either hemisphere. In view of the almost complete return of voluntary power after the previous paralysis, contrasted with the difficulty of eliciting even slight movements by electrical stimulation of the affected cerebral cortex, it is evident that there must be an essential difference between the elicitation of movements by volition, and by electrical excitation of the cerebral cortex.

It must be pointed out that in this animal only that portion of the cortico-spinal tract that runs in the right lateral column (the crossed pyramidal tract) was divided; the uncrossed tracts of the ventral columns escaped, and as they were not degenerated we have no means of estimating what proportion of the cortico-spinal fibres they contained. Grünbaum and Sherrington have shown that the ventral pyramidal bundles are present and as variable in the higher apes as they are in man; as they do not exist in monkeys or in animals lower in the scale, reference to them was not necessary in our accounts of those animals.

In this chimpanzee, as in our other animals after spinal lesions, reactionary chromatolysis could be detected only in the giant pyramidal cells of the cerebral cortex, and they were almost entirely limited to the hemisphere of the side opposite to the spinal lesion. We searched carefully for similar changes in other cells, but though a few of the ordinary large pyramidal cells presented nuclei displaced towards the periphery, and occasionally a partial absence of tigroid bodies in the neighbourhood of the nuclei, we could not convince ourselves that there was here any definite chromatolysis. It may be noted that the giant-cells of the chimpanzee brain are of very various sizes, some of them scarcely exceeding in bulk the ordinary large pyramidal cells; but though none of the latter were affected, changes could be seen in giant-cells of all sizes. This fact is of interest, for, as will be shown later, it is these giant-cells which give origin to the cortico-spinal fibres, and they vary considerably in calibre in the spinal cord.

Careful enumerations of the giant-cells of each hemisphere were made, and on comparison it was found that those of the left cortex were considerably reduced in number, to the proportion of about four to seven as compared with the opposite side. Further, the interesting fact came out that while in the leg and body areas the giant-cells in chromatolysis were about four times as numerous as the unaltered cells, in the forearm and hand areas the proportion of the affected to the unaffected giant-cells was as seven to one. This fact may be possibly correlated with the greater bilateral representation of the lower limb and trunk movements, but as the direct ventral pyramidal tracts were not divided, it seems that in the upper cervical segments of the cord the cortico-spinal fibres which subserve the leg and body are relatively less numerously represented in the lateral (crossed) pyramidal tract than those subserving the forelimb, whereas in the direct pyramidal tracts the converse obtains.

The area delimited by these affected cells is indicated in fig. 7. It lay immediately in front of the fissure of Rolando. On the mesial surface of the hemisphere it assumed the shape of a flattened inverted pyramid which just extended down to the callosomarginal sulcus. On the lateral surface it was limited posteriorly by the characteristic structure of the posterior central gyrus, which, in the upper part of the hemisphere, coincided almost exactly with the floor of the central fissure; but, more laterally, the affected area receded more and more from the floor of this fissure—that is, it ascended the anterior wall of the sulcus, in proportion with the increased distance from the middle line. In the many hundreds of sections examined in this animal not a single affected cell was found posterior to the floor of the fissure of Rolando. In the upper part of the lateral surface the area included the whole of the precentral gyrus and a considerable portion of the frontal lobe immediately anterior to it, but its lower part extended only just across the anterior central gyrus.

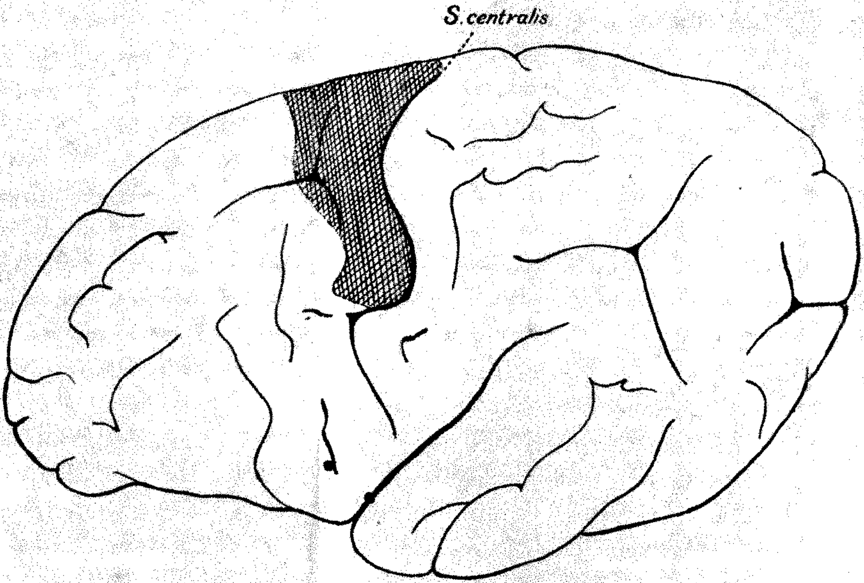

FIG. 7.

The lateral surface of the brain of a chimpanzee showing the region in which cortical changes were found after section of the cortico-spinal fibres in the right side of the spinal cord.

The cell-changes cease almost abruptly just above the inferior genu of the central fissure, for in sections immediately below this not more than one in every fifty cells was affected, and even these disappeared in the space of 2 mm. or 3 mm.

Human Cases.

Although the cortex of the Rolandic region of the brain has been systematically examined by one of us (G. H.) in a large number of cases in which the pyramidal tracts had been involved in local lesions or system degenerations of the spinal cord, the only material of which we shall make use in our present communication was obtained from two cases in which there was complete or almost complete destruction of these tracts by traumatic lesions of sudden onset. By excluding cases in which the disease was slowly progressive, and the interruption of the fibres therefore not sudden, we are in less danger of adopting conclusions not warranted by facts.

Case 1.—B. C., aged 17 years, was admitted to St. Bartholomew's Hospital under the care of Mr. W. Bruce Clarke, to whom we are indebted for permission to use the case, with a fracture-dislocation of the vertebral column in the lowest cervical region. The injury was received in a lift accident. After recovery from the shock it was ascertained that there was complete loss of all forms of sensation below the cutaneous distribution of the seventh cervical roots, with complete paralysis of the lower extremities and trunk as well as of the movements of the fingers and wrists. The shoulder movements were unaffected. Flexion, supination and pronation of the elbows were possible, but were considerably weaker in the left than in the right limb; the right elbow could be extended very weakly, but on the left side this movement was completely paralysed. Death occurred 229 days after the accident.

Dr. Hinds Howell, who examined the spinal cord, has kindly given us the following report: "There was a complete transverse lesion of the cord in the seventh cervical segment; no fibres pass through this level intact. The lesion extends higher on the left than on the right side reaching the lowest part of the sixth segment. Such a lesion naturally produced total degeneration of the pyramidal tracts below its level."

The brain was received hardened in formalin. The hemispheres were photographed and the whole of the Rolandic region of each, with the paracentral lobules, was divided into suitable blocks, the position of each block being carefully marked on the photographs. The sections obtained from each block could be thus accurately localized. They were stained with thionin.

Identical changes were found in both hemispheres. In the paracentral lobules in front of the upper extremity of the fissure of Rolando there was a practically complete disappearance of the large giant-cells which characterize this region; in numbers of serial sections not a single one could be seen, and on the right side in which all present were counted there were not more than two or three in every twenty sections,

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and almost all of these were degenerated; the normal average number of giant-cells per section in this region is about thirty. The majority of the cells had disappeared without leaving a trace of their previous existence, but here and there masses of pigment, or small clumps of neurophages, or of glial nuclei marked the positions they had occupied. Despite this extraordinarily severe affection of the giant-cells of the infra-granular layer we could detect no pathological change in, or numerical reduction of, the ordinary large pyramidal cells of the infra-granular or supra-granular layers; the polymorphic layer and the layers of small and medium-sized pyramids also appear to have escaped entirely. The extent of the area in the paracentral lobules which shows this change is represented in fig. 8.

The same pathological condition was found on the lateral surface of the brain near the apex, but about 15 mm. from the latter, measured along the fissure of Rolando, giant-cells begin to reappear, at first in very small numbers, but their number increased gradually lateralwards. The majority of these cells, however, were far from normal; some were in the advanced stages of chromatolysis, others were represented only by atrophied and shrunken remains. At about 20 mm. from the apex, still above the level of the superior frontal sulcus, there was in the space of a few millimetres a great increase in the number of normal cells, and degenerated and atrophied ones disappeared from the sections. The transition of the abnormal to the normal cortex can be scarcely described as abrupt, as through the space of a few millimetres normal and degenerated cells were found together, but it could be certainly easily defined.

Immediately below this area there came a strip of cortex on the anterior wall of the fissure of Rolando and on the anterior central gyrus, the structure of which was normal; it contained the standard number of giant-cells. But at a level which was approximately opposite the middle of the second frontal convolution the giant-cells again became less numerous than normal, and many of them were atrophied or in chromatolysis. The transition here between the cortex which was normal and that which was affected was, however, quite gradual; and the pathological changes in this area were not so marked as in the paracentral lobule and the upper third of the anterior central gyrus. The giant-cells were nowhere reduced to less than a third of their normal number, and a certain proportion of them were intact. The extent of this area of pathological cortex is represented in fig. 8; its lower margin could be more easily delimited than its upper, but it was by no means sharp. The distribution of these changes along the fissures of Rolando was the same on the two sides.

The topography of this affected area on the lateral surfaces of

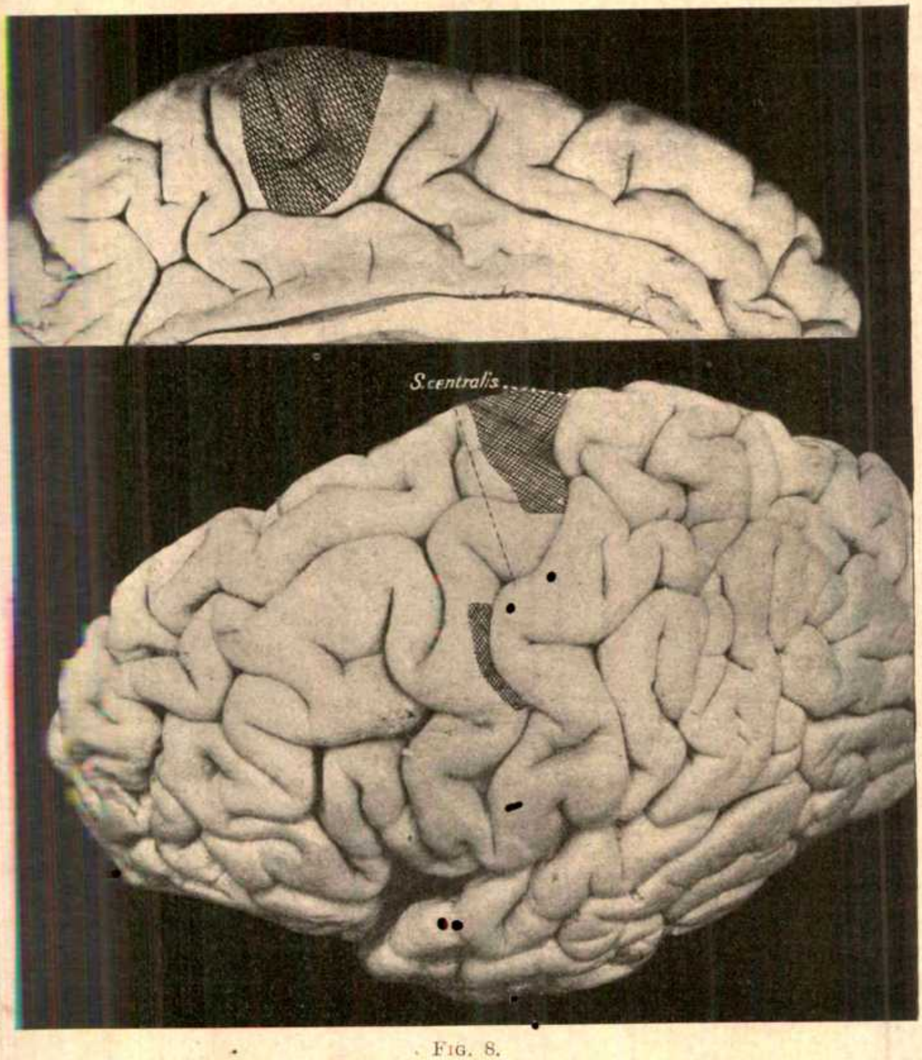

FIG. 8.

Photographs of the brain of Case 1. The shaded areas represent the regions in the surface of the brain in which cell-changes were found.

ispheres may be briefly described. Its posterior margin coin- lly everywhere with the bottom of the fissure, but it ich resemble the "Betz" cells in size, but lie within the oral limit of the post-central cortex—in the "transition area" of Brodmann—were not affected. The anterior limit of the area is most easily understood from our illustration; it must be, however, added that in the lower area the greater part of the altered cortex was hidden in the fissure of Rolando, and only a relatively small portion of it appeared on the convexity of the anterior central gyrus.

Absolutely no histological changes could be detected posterior to the fissure of Rolando, in front of the frontal edge of our areas, or in any other region of the cerebral cortex.

Case 2.—A woman, aged 68, suddenly lost the power of movement in her lower limbs and hands after a fall on her back. On examination a few days after the accident there was complete loss of power in the lower extremities and trunk muscles, as well as incontinence of urine and fæces. The movements of the shoulder-joints were unaffected, and flexion of the elbows was possible, though weak; there was almost complete paralysis of the extensors of the elbows and of all the movements of the wrists and fingers. This condition improved very little before death, which occurred 108 days after the accident. We are indebted to Dr. J. R. Lunn for this case.

The spinal lesion was found on *post-mortem* examination between the sixth and seventh cervical segments at practically the same level as that in Case 1. The dorsal columns, the dorsal horns, and the whole of the right lateral column excepting its periphery were completely destroyed at this level, and the lesion also extended into the left lateral column probably far enough to interrupt almost the whole lateral pyramidal tract. On examination of the cord by Marchi's method there seemed to be below the lesion complete degeneration of the right crossed pyramidal tract, but the degeneration was not so great in the left lateral column. As the lesion was limited to the dorsal half of the cord the direct pyramidal tracts of the ventral columns escaped, and we had no means of estimating the proportion of the cortico-spinal fibres these carried in this case.

The examination of the cortex was conducted in the usual way. The description of the changes we found may be limited to the left hemisphere, as they were more pronounced on this side than on the right, but were similar in both except in degree.

The normal giant-cells were counted in series of sections fr
every 2 or 3 mm. along the f[illegible] of Rolando and the
per section at

with the numbers from the corresponding levels of normal hemispheres, gave a fair indication of the relative affection of the cortex at different levels. In the paracentral lobule the affected area lay entirely in front of the upper extremity of the fissure of Rolando; it contained only about 2·5 intact giant-cells per section, while the normal average is about 30. In every section large numbers of giant-cells in chromatolysis could be seen, and a striking feature was the great excess of pigment all the affected cells contained. This pigment was present in unusual amounts in many of the giant-cells which have been included in the normal count, as considering the age at which the patient died its presence, unless associated with disintegration of the tigroid and displacement of the nuclei, was not regarded as evidence of degeneration. Often only masses of pigment without any stainable cytoplasm or a nucleus represented degenerated cells, and not infrequently clumps of neurophages laden with such pigment could be seen. Only the giant-cells of the infra-granular layer were affected, and it is noteworthy that there was no great excess of pigment in the other cells of the cortex.

On the lateral surfaces of the hemispheres the areas of affected cortex were practically identical with those mapped out in Case 1. From the mesial margin to about the middle of the superior frontal gyrus the relative reduction of the giant-cells was practically the same as in the paracentral lobule, but here a transition area appeared which contained both normal and pathological cells, the proportion of the former increasing as sections were examined from above downwards. A short distance above the level of the superior frontal sulcus chromatolytic and atrophic cells disappeared, and an area of normal cortex covered the precentral gyrus to just below the inferior genu of the fissure of Rolando. From here for a distance of about 15 mm. along the fissure there was another definitely abnormal area of cortex in which the giant-cells were considerably diminished—to about half of the normal—and many which persisted were in chromatolysis. The topography of this area was practically the same as in Case 1, and, as in that specimen, the transition from the normal to the abnormal cortex was not by any means abrupt or sharp; consequently a definite line could not be drawn around the area. The cortex of the lower quarter of the anterior central gyrus was intact.

The posterior limit of the affected areas on the lateral surfaces of the hemispheres fell along the bottom of the fissure of Rolando; the anterior limit can be best understood from fig. 8. From this it is evident that the greater part of the lower area lay in the anterior wall of the fissure of Rolando. Absolutely no changes could be detected in the cortex behind this fissure, or in any other region of the brain.

The Cortical Changes which Result from Capsular Lesions.

A short description may be given here of the cortical changes which were found in cases in which the internal capsule as well as part of the central ganglia had been destroyed some time before death, but as lesions in this region must interrupt, in addition to the pyramidal tracts, projection-fibres from other regions of the cortex the facts we have obtained from these cases have less direct value for the problems we attempt to solve.

Case 3.—A female, aged 26, died six weeks after the onset of a right hemiplegia, which was due to vascular embolism and a softening which destroyed the left internal capsule, and almost the whole of the nucleus lenticularis and of the optic thalamus.

The cortical changes were of interest, especially in contrast to those we found secondary to spinal lesions. Throughout the whole of the left anterior central gyrus and the paracentral lobule there was a considerable diminution of the giant-cells which characterize this region, and very few of those persisting were normal. The majority were in chromatolysis or typical *reaction à distance;* the swelling of the cells and the excentric position of the nuclei seemed to be a more prominent feature than in the reaction of these cells to spinal lesions. The disintegration of the tigroid bodies was complete in most of the affected cells, in many the dendrites were shrinking or had disappeared, and some cells were already represented only by masses of pigment.

These changes were not, however, confined to the giant-cells or to the areas in which we detected cortical cell-changes after spinal lesions. In addition to the giant-cells many of the ordinary large pyramidal cells of the infra-granular layer, and a smaller proportion of the supra-granular or superficial pyramids, were similarly affected, and it could be ascertained beyond doubt that a considerable proportion of the former had disappeared. No changes could be detected in the more superficial layers of the cortex, or in the layer of polymorphic cells.

Further, these changes were not limited to the precentral area; in the posterior central gyrus, and indeed in all parts of the cortex which were examined, a not inconsiderable proportion of the infra-granular pyramids and a smaller proportion of the superficial pyramids were in chromatolysis, and in most regions it appeared probable that many of the larger cells of the deeper layer had disappeared. Unfortunately the whole of the cortex of this hemisphere was not systematically examined.

This affection of the cortical cells can scarcely have been a direct result of the vascular lesions, as the softening did not extend into the cortex, and it was quite as prominent in the paracentral lobule and at the apex of the hemisphere, both of which receive their blood-supply from the anterior cerebral artery, as in the area of distribution of the middle cerebral artery.

Case 4.—A man, aged 41, had a stroke producing a right hemiplegia about eighteen months before his death. Part of the left internal capsule was destroyed with portions of the central ganglia; the degeneration of the cortico-spinal fibres was so complete that the left bulbar pyramid was practically devoid of myelinated fibres.

The chief cortical changes were: (1) almost complete absence of giant-cells from the precentral cortex; (2) a definite loss of the ordinary large pyramidal cells of the infra-granular layer in both the precentral and the postcentral cortex, with increase of neuroglia throughout this layer; and (3) possibly some cell-defect in the superficial layer of large pyramidal cells, but on this it was extremely difficult to decide with certainty.

A short summary of our observations may be given in a few sentences. By making use of the reactionary chromatolysis and the subsequent changes which occur in nerve-cells after division of their axis-cylinders, we have been able to determine the cortical cells which react to section of the cortico-spinal tracts, and from which we must assume, therefore, that these fibres take origin in man, the chimpanzee, certain monkeys, the lemur, and in representative carnivora.

In the primates (man, ape, and monkey) the area which contained these cells lay entirely in front of the sulcus centralis; in the carnivora it was in less close relation to any sulcus, but it was chiefly behind the sulcus cruciatus.

Further, the only cells from which we have been able to show that cortico-spinal fibres spring are the giant-cells of the infra-granular layer, which characterize this region of the cortex in all the mammals we have used in this investigation.

Before discussing the significance of these observations, and the conclusions that may be drawn from them, it is advisable to give a short review of the more important contributions to our subject that have been already published. But very few of these deal directly with the origin of the pyramidal tracts.

Several authors have directed attention to the alterations of the cells of the cerebral cortex which follow lesions of the internal capsule. von Monakow [20] writes that "all the giant-cells (Riesenpyramiden) in both man and animals degenerate after the destruction of the internal capsule," and "the pyramidal tracts take origin from the giant-cells of the central convolutions" (S. 198); but there can be no doubt, from his text and his illustrations (*see* his fig. 113), that in this connection he applies the term "Riesenpyramiden" to the large cells of the supragranular layer; in fact, the giant-cells of Betz are not found in both central convolutions, but only in the anterior. Dotto and Pusateri [9] have also observed degeneration of the cortical pyramidal cells after destruction of the internal capsule; and Marinesco [17] has recorded the cortical changes he found in six cases associated with capsular lesions of variable duration. These alterations included chromatolysis, atrophy, or disappearance of the large pyramidal-cells, dependent on the duration of the lesion before death. It is not, however, evident from his paper if it was only the large giant- (Betz) cells that he saw affected, and he has not described definitely the area in which these changes were present. At about the same time Ballet and Faure [2] demonstrated the *reaction à distance* in the large cortical pyramidal-cells in dogs after subcortical section of the projection-fibres; and more recently Gentès and Bellot [12] have studied the alterations in the neurofibrils of these cells that occur secondary to destruction of the internal capsule in man. In neither of the last-mentioned papers is the region in which these reactionary changes were found, or the cells which were affected, accurately described. But, despite the intrinsic interest of these observations, it is evident that no conclusions as to the origin of the pyramidal tracts can be drawn from them, as when these are completely destroyed in the internal capsule many of the projection-fibres which pass from the cerebral cortex, as well as the cortico-pontine tracts, must be also interrupted, and the cells from which these take origin would consequently show the same changes as those from which the cortico-spinal fibres arise.

In a later communication Marinesco [18] has described the cortical changes he discovered in six cases of paraplegia due to local spinal lesions. The cell-changes were described as limited to the paracentral lobule and the upper third of the anterior central gyrus, and only the giant-cells of this region were affected. The chromatolytic changes supervened later and were slower in their course than those which resulted from interruption of the fibres of the internal capsule. It is interesting that Rothmann [24], in the discussion which followed the presentation of Marinesco's paper, stated that four weeks after transverse section of a dog's spinal cord "the cortical cells were completely normal"; but it is not clear if attention was specially directed to these, or what parts of the cortex were examined.

The first deliberate attempt to define the exact origin of the corticospinal tracts has been made by Campbell [7] in connection with his work on the histological localization of cerebral function. This author came to the conclusion at which we have arrived, in fact he regarded "the Betz cells as discharging elements for motor impulses," but the arguments upon which he relied were scarcely sufficient to support this conclusion. In the first place, he made use of the cortical changes he found in two cases of amyotrophic lateral sclerosis; he writes of this as "a disease limited in its affection to the muscular system and the motor systems of neurones," and consequently argues that the fact that the cortical changes he discovered were limited to the precentral area "constitutes the strongest confirmatory evidence yet adduced to support the assumption that in man, as well as in the anthropoid apes, the elements acting for the direct control of muscular movements are confined to the precentral gyrus and its paracentral annexe." But, unhappily, his premise is absolutely inaccurate, as amyotrophic lateral sclerosis, as is now well known, is not a disease limited to the motor systems of neurones—in fact, in a series of ten cases examined by one of us (G. H.) there was invariably only some degeneration of the spino-cerebellar tracts, in the peduncles of the cerebellum, and in several tracts of the brain-stem, which cannot be regarded as efferent or "motor." Further, as Campbell himself rightly recognized, the cortical disease is not limited to the giant Betz cells, for there is also a "general cell-disturbance," and especially atrophy and disappearance of the supra-granular pyramids; the conclusion, therefore, he has tacitly drawn, that only the Betz cells give origin to pyramidal fibres, is manifestly unjustified.

Further arguments in support of his conclusion were obtained from the examination of brains of patients who died at long intervals after the amputation of parts of limbs. In these cases the chief cortical change was a condition of the giant-cells identical with the *reaction à distance*, and this despite the fact that the amputation to which it is attributed was in some of the cases of several years' duration. Similar changes had been previously described by Acquisto and Pusateri [1] and by Peli. The interpretation Campbell has placed on them may be questioned. In the first place, the problem arises: Are these chromatolytic changes to be ascribed to a retrograde atrophy which oversteps the anterior cornual synapses, and thus involves the upper motor neurones? That this really occurs seems doubtful, as permanent atrophy of even the anterior horn-cells is far from constant after amputations. Further, the cytological changes Campbell describes are certainly not such as might be expected to result from this condition, and it is noteworthy that he could detect no diminution of the affected cells, though on his own showing the cell-changes must have existed for years. There is another possibility which Campbell himself seems to have considered in raising the question "whether, in the case of the cortical motor element, suppression of the discharge of energy which it elaborates, by division of the second link in the neuronic system," can "result in corporeal disintegration"; it is that the disturbance of the physiological equilibrium of the motor cortex which must result from the amputation of a limb may lead to structural change in its cells. If this be so, Campbell's conclusion as to the cells of origin of the pyramidal tracts receives little support of any value, for besides the fact that it is unsatisfactory to argue from the physiological to the anatomical, it cannot be maintained that changes resulting from such physiological inactivity will express themselves only on the cortico-spinal neurones; it seems more probable that the whole system of neurones concerned in the elaboration of the cortici-fugal discharges would be involved. In fact, Campbell invalidates his chief conclusion by the statement, "particular attention was not paid to the other cell-layers; and although we think that the large pyramidal cells had suffered a reduction in numerical strength, we cannot attach much importance to the observation, because we did not verify it by making careful enumerations and drawings."

IV.

The first question with which we have to deal is: *From what region of the cerebral cortex do the cortico-spinal tracts take origin?*

It must be at once pointed out that in the present communication we are concerned only with the actual cortico-spinal fibres—that is, those which form the upper motor tract for the volitional movements of the limbs and trunk—and that we cannot deal with the fibres which pass from the cortical centres for the movements of the eyes, or to the bulbar lower motor centres. This restriction has been necessary owing to the method on which we have relied, as on dividing the pyramidal tracts above the spinal cord other cortici-fugal fibres would be also injured, and this would seriously complicate our task, and might invalidate our conclusions.

The region of the cerebral cortex from which, according to our observations, the fibres of the cortico-spinal tracts take origin, has been already mapped out in each of the brains we have examined; further reference is therefore necessary only to the exact topographical relation of this area in each class of animals to the more constant cerebral sulci.

In the macaque monkey it lay entirely in front of the fissure of Rolando and never extended to the posterior central gyrus, but the exact relation of its posterior margin to the floor of the sulcus was variable, as towards the apex of the hemisphere it practically coincided with it, but more laterally it gradually receded from the floor along the anterior wall of the fissure. A similar relation was observed in the chimpanzee.

In the two human brains we have used in this work the posterior limit of the area from which we have shown that the cortico-spinal fibres take origin never crossed the bottom of the sulcus, but in a few of the many hemispheres, normal and pathological, in which this region has been examined by one of us, the giant-cells from which we shall show that the cortico-spinal fibres spring extended behind the fissure, and in one case almost reached the apex of the posterior central gyrus. In fact, as Schäfer, Sherrington [26], Elliot Smith, Vogt [29], and Brodmann [5] have insisted, the relation of even the chief fissures to homologous cortical areas is by no means constant even in the same species, and their importance in cortical localization may be consequently overrated.

It is noteworthy that the region of the cerebral cortex that gives origin to the cortico-spinal fibres subserving the trunk and limbs in both the dog and cat lies chiefly behind the sulcus cruciatus, and does not extend lateral to the sulcus coronalis.

In the lemur none of the margins of the area can be accurately defined by the position of a sulcus.

There are very few anatomical observations known to us which help to confirm our conclusion that in the primates the cortico-spinal fibres spring only from the cortex in front of the sulcus centralis, though it may be seen from the extracts in our opening paragraphs that there is a tendency to the opinion that the major part of these bundles come from the anterior central gyrus.

Campbell's [7] work, and especially his arguments based on the cortical changes he found in amyotrophic lateral sclerosis and after amputations, are extremely suggestive, but, as we have seen, his conclusions are open to serious criticism.

Mellus [19], however, failed to trace degenerated fibres into the spinal cord by the Marchi method in several macaque monkeys after the removal of Beevor and Horsley's "thumb centre" in the posterior central gyrus, and only in one animal could he demonstrate a few. Further, Grünbaum and Sherrington [14], working with the anthropoid apes, were unable to find any degeneration in the spinal cord after removal of portions of the cortex of the posterior central gyrus, though spinal degeneration was always obtained from lesions of the anterior central gyrus.

On the other hand, Vogt [29] found, besides extensive degeneration of the pyramidal tracts after destruction of the anterior central gyrus very slight ("sehr geringfügige") pyramidal degeneration from lesions of the posterior central cortex; unhappily, a detailed description of his observations is not given. Von Monakow, too [20], from the investigation of secondary degeneration from cortical lesions, came to the conclusion that in man pyramidal fibres spring from the posterior central gyrus.

But evidently after cortical ablations a negative result is of much greater value than a positive; and it seems to us that a point of fundamental importance has been definitely settled in the present investigation carried out over a long series of animals, and in man by a method free from the fallacies which so easily arise in the use of the excitation method, which indeed have given such contradictory results in even the best hands, viz., that *the posterior central (ascending parietal) gyrus is in no way directly concerned with the origin of the motor (pyramidal) fibres subserving the limbs and trunk.*

V.

The second question with which we are concerned is: *From what cells of the cerebral cortex do the cortico-spinal fibres take origin?*

In all our material in which the cortico-spinal tracts had been destroyed by lesions so situated that no other cortici-fugal fibres could have been injured the reactionary cell-changes could be found only in the infra-granular layer of the cortex (Campbell's VI., Brodmann's V. layer); we never succeeded in detecting chromatolytic changes in the superficial pyramids, in the polymorphous layer, or in any of the smaller pyramids or granule cells. The infra-granular layer of the area of cortex to which we found the changes limited contains not only large pyramidal cells which correspond approximately in size and shape to those which occupy this layer in other regions of the cortex, but also cells much larger, more bulky or pyriform in shape, more richly supplied with coarse masses of chromatophilic material, and possessing a nucleus which is small in proportion to the cell-body. These giant-cells, which were originally described by Betz, and later by Bevan Lewis and Clarke as the "ganglionic cells," are very variable in size; some, indeed, scarcely exceed in bulk those which we regard as ordinary large pyramidal cells, but from these they may be easily distinguished by their shape and staining reactions. It has been in these giant-cells, and in them only, that we have found changes when the cortico-spinal tracts alone of the cortici-fugal fibres were destroyed by the lesion. We must therefore conclude that it is from these cells, and from them alone, that the pyramidal tracts spring.

The question then naturally arises: Do these giant-cells which are limited to the region from which the pyramidal tracts arise give origin only to cortico-spinal fibres? It is difficult to give a definite answer to this, but it appears to us that the reply should be in the affirmative. After experimental section of one pyramidal tract in the higher cervical segments of the cord we always found, even after considerable periods, a certain proportion of the giant-cells of the opposite hemisphere remaining intact—in the callithrix monkey, for instance, about one-twelfth to one-fifteenth of the normal number. But this is what should be expected even if all the giant-cells do send their axis-cylinders to the spinal cord, as in all mammals a certain proportion of the cortico-spinal fibres run through the homo-lateral columns of the cord and consequently escape injury in a lesion of the opposite side. In this fact, too, we find the explanation of our observations that faradic unipolar stimulation of the affected cortical area of the left hemisphere in this monkey produced only movements of the limbs of the same side.

But this explanation of the escape of a certain number of the giant-cells in the hemisphere on the side opposite to the spinal lesion demands, if it be correct, that a certain proportion of those in the corresponding regions of the homo-lateral hemisphere, approximately equal to that remaining intact in the opposite, should be affected. And in fact we have found this to be the case in every one of the brains we examined within the two months after unilateral section of the spinal cord; it would be evidently scarcely possible to make this observation at the stage in which the only prominent change in the cortex was disappearance of the affected cells, as it was in one of our monkeys.

In our human *Case* 1, on the other hand, in which there was a complete transverse lesion of the spinal cord in the seventh cervical segment, scarcely a single normal giant-cell could be found in either paracentral lobule, or in the apical portion of the anterior central gyrus; that a certain proportion remained intact at the lower margin of the affected area on the lateral convexity and in the region of the cortical motor-centres for the finger and hand movements, may be explained partly by assuming a certain overlapping of the centres with normal and interrupted projection systems, and partly also from the fact that the plane of our sections was not exactly parallel to the lower margin of the affected area. On the other hand, the escape or slighter affection of some of the cells may have been possibly due to the existence of collaterals from their axis-cylinders to the corpus callosum or elsewhere, so that sufficient function was still possible to the cells to save them from degenerative changes.

From the above facts we may conclude that certainly almost all, if not all, the giant-cells of the portions of the precentral areas we have investigated send their axis-cylinders to their cortico-spinal tracts.

The published observations we can collect to corroborate this view are few, though it has been several times already brought forward, but without any unequivocal proof or even strong evidence in support of it. Cajal [6] indeed, who assumed that the pyramidal tracts spring from the large pyramidal cells of the fourth and sixth cortical layers, immediately adds, "but we must remark that this is only a more or less plausible hypothesis which is entirely without physiological and anatomo-pathological foundation."

According to von Monakow [20], "the pyramidal tracts take origin from the giant pyramidal elements (Riesenpyramidenkörpern) of the central convolutions"; but as has been already pointed out, the large superficial pyramidal cells are referred to here; indeed, it cannot be the giant- (Betz) cells alone, as these exist only in the anterior central gyrus. It appears to us that of the other authors, Marinesco alone had, in the cortical changes he observed secondary to spinal lesions, sufficient evidence for any definite statement as to the cells of origin of the cortico-spinal tracts.

The changes which develop in the precentral cortex secondary to capsular lesions, and those we found in the lemur after unilateral section of the crus, may be contrasted here with those which result from destruction of the cortico-spinal fibres alone. In the latter condition we found only the giant-cells affected, while after interruption of the projection fibres from the cortex to the central ganglia and lower centres by capsular lesions or by section of the crus there occurs in addition chromatolysis, and later atrophy and disappearance, of many of the pyramidal cells of the supra- and infra-granular layers, but especially of the latter. Von Monakow also found the large pyramidal cells degenerated after capsular lesions, but it is not evident from the descriptions given by Marinesco, and by Ballet and Faure, whether they observed changes in these ordinary large pyramidal cells or only in the giant-cells.

It therefore appears that, in the central gyri at least, the ordinary large pyramidal cells, but especially those of the infra-granular layer, give origin to the shorter projection fibres of the cortex. And the giant-cells may be regarded as large pyramidal elements modified in the course of phylogenesis as the cerebral cortex acquires a direct connection with the spinal cord, and we consider from data obtained in the present research that *the infra-granular giant pyramidal cells undoubtedly give origin to the cortico-spinal fibres subserving the limbs and trunk, and in the cortical areas under consideration to these only.*

VI.

The question as to *whether the origin of the cortico-spinal tracts is co-extensive with the excitable cortical area subserving the limbs and trunk* is of great interest, but a satisfactory reply is rendered more than usually difficult by the fact that "the excitable motor cortex" is a very relative term, and the areas to which it has been applied have been mapped out strikingly differently by various observers; of this anyone can convince himself by a glance at Plate 4 of Vogt's [30] monograph, in which the results obtained by various experimenters on the cercopithecinæ are reproduced.

Evidently the extent of cortex from which movements can be obtained depends on several conditions, and even when minimal stimuli are employed the state of anæsthesia, the condition of the exposed brain, and factors which facilitate the spread or irradiation of the current both superficially and deeply can seriously influence the results. These complicating factors have made the term "motor cortex" very vague and indefinite.

From the anatomical point of view "the motor zone would be best and most sharply defined and delimited as the region of the pyramidal tracts" (v. Monakow), but anatomy has failed hitherto to delimit this region accurately. We may, however, attempt to show how closely the areas we have mapped out by anatomical methods coincide with the "motor cortex" as defined by the more recent physiological investigations.

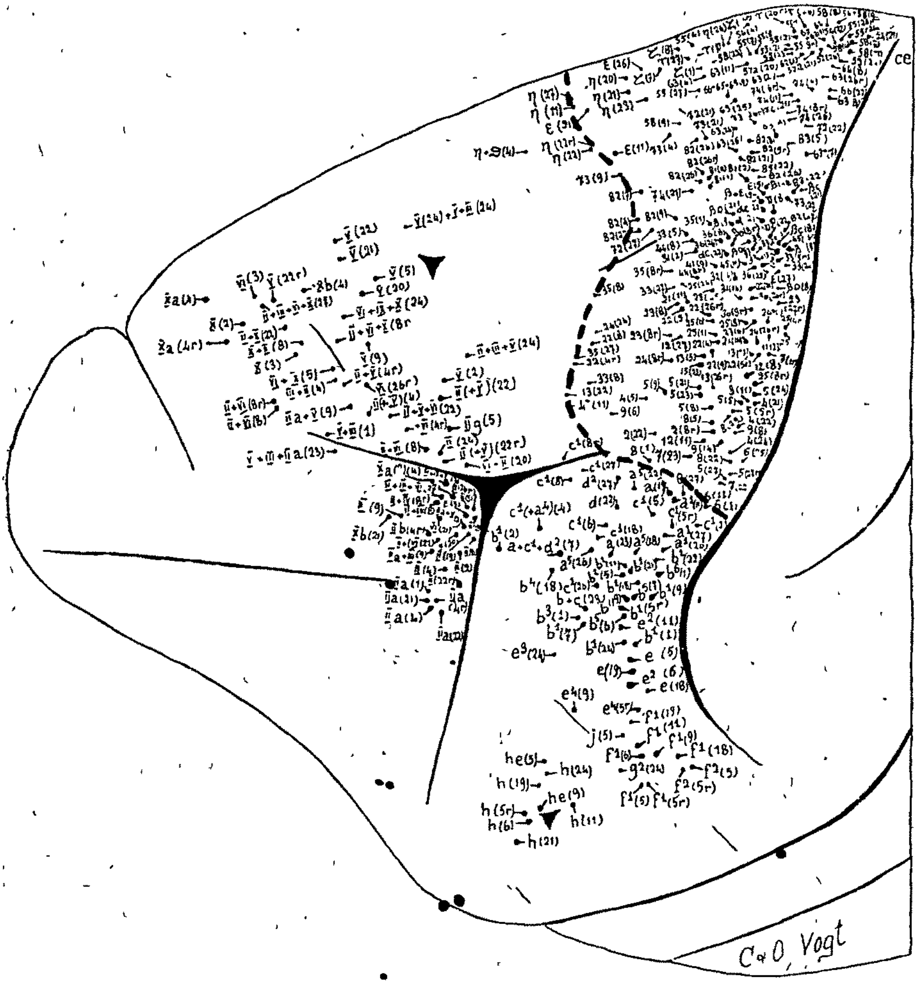

FIG. 9.

A reproduction of Vogt's figure showing the cortical centres for primary movement in the lateral surface of the hemisphere. The centres for the movements of the limbs and trunk are enclosed in front and below by the thick broken line.

In no other class, perhaps, have the excitable cortical centres been so frequently or so carefully studied as in the lower monkeys, especially in the macaque family, and in this class consequently we may begin our attempt to correlate our anatomical facts with physiological conclusions. For this purpose we select the work of C. and O. Vogt, who recently carried out experiments in a large number of animals with every possible precaution [30].

Fig. 9 is a reproduction of Vogt's composite diagram of the results obtained by unipolar faradic stimulation of the cortex in twenty-seven animals, the centres for the movements of the limbs and trunk being enclosed by a thick line. On comparing this with our fig. 4 obtained by an entirely different method the practical identity of the area of origin of the cortico-spinal fibres with this portion of the "excitable motor cortex" is very striking. The absence of excitable foci in the posterior central gyrus, and the close correspondence between the line in Vogt's diagram which separates the centres for the upper limb from those for the facial movements, with that of the lower limit we obtained for the area of origin of the cortico-spinal fibres, are worthy of note. The frontal margins of the two areas also correspond fairly closely, except perhaps that our area includes part of Vogt's "unerregbareres präcentrales Feld," that is, a small area lying above and in front of the superior precentral sulcus. On the other hand, Vogt places no excitable foci in the gyrus marginalis on the mesial surface of the hemisphere, while we have found that a not inconsiderable proportion of the cells of origin of the cortico-spinal tracts lie in this region. It must be, however, pointed out that Vogt's observations on the inexcitability of the mesial surface of the hemisphere are at variance with those obtained by many other investigators, especially Horsley and Schäfer; the German authors, it is true, obtained movements by stimulation of this region, but came to the conclusion that it is not an excitable area in the restricted significance of the term, as identical movements could be always obtained from the lateral surface of the hemisphere, and stronger currents were always needed than there; further, the movements obtained from the mesial surface were more variable and less localized than those from the convexity of the brain. Further physiological investigations may be awaited before any attempt is made to explain this discrepancy between the physiological "excitable cortex" and the region of origin of the cortico-spinal fibres in the gyrus marginalis. It is noteworthy that in the orang, C. and O. Vogt discovered true excitable foci on the mesial surface, as Sherrington and Grünbaum had previously done in the chimpanzee.

Records of stimulation experiments on the cerebral cortex of the lemur have been recently published by Völsch [31], O. and C. Vogt [30], and by Mott and Halliburton [21]; previous observations had been made by Elliot Smith and Page May [27], and again by Elliot Smith and W. H. Wilson [28]. One of us (W. P. M.) has during the last three years made similar observations on six other lemurs, and these excitation experiments, which will be published in detail later, showed definitely that, as in the post-central gyrus of the monkeys and apes, no motor response could be produced by stimulation with a minimal strength of current behind the sulcus X, though when an electrode was placed in its depth movements, most frequently of the hip, could be obtained. In fact, the posterior limit of the excitable cortex coincides almost exactly with that of the area mapped out in fig. 3, not only on the lateral surface but on the mesial as well, and indeed the extent of each area is almost identical. These excitation results agree with those obtained by one of us in conjunction with Elliot Smith five years ago, and more recently by C. and O. Vogt, but the latter do not extend the area to the mesial surface of the hemisphere. On the other hand, Halliburton and Mott have mapped out the motor centres over a much more extensive area of cortex, especially posterior to sulcus X.

It may be pointed out that one lemur was the only animal in which the upper motor neurone fibres for the face, tongue and head movements were divided, and corresponding thereto we have in this animal alone found histological changes in the cortical centres for these movements.

The excitable "motor" cortex of the chimpanzee has been mapped out by Grünbaum and Sherrington [13] in their classical contributions which form a landmark in cerebral localization. The stimulation results obtained in our animal entirely confirm their conclusions, but with us the area, stimulation of which by minimal currents gave motor responses, extended slightly in front of the precentral sulcus; for instance, in the right hemisphere flexion of the wrist was obtained 4 to 5 mm. in front of it. In fact, the anterior limit of the excitable area, as defined by physiological methods in the right hemisphere, coincided exactly with the frontal margin of the area of origin of the pyramidal tracts, as delimited by anatomical methods in the left hemisphere.

The slight discrepancy which exists between our histological results and the physiological results of Grünbaum and Sherrington may be due to individual variation; in fact, these authors have insisted on the inconstancy of the sulci, and therefore on the fallacy of using them as strict landmarks. On the other hand, as Campbell has already pointed out, and we confirm the observation, the giant-cells become sparser towards the frontal margin of the area, and this region must therefore, though part of the motor cortex, give a smaller response to the same strength of current than would be obtained more posteriorly. Grünbaum and Sherrington recognized this fact in their stimulation experiments, for they write that "the anterior limit of the motor field is not of a sharp, abrupt character, but fades off forwards somewhat gradually. This edge extends further forward under *Bahnung*." It is therefore evident that in the chimpanzee, too, the origin of the cortico-spinal tracts is closely co-extensive with the excitable "motor" cortex for the limbs and trunk.

It is useless to attempt to discuss in detail the topographical relation of the area of origin of the cortico-spinal tracts in man to the excitable motor cortex, as the latter naturally cannot be investigated by surgeons under such favourable conditions as in the lower animals. Yet a considerable number of observations, especially by Horsley, Cushing and Krause, have accumulated, from which it appears extremely probable that in man, as in the anthropoid apes, the motor centres lie entirely in front of the fissure of Rolando, and occupy only a relatively small area on the surface of the anterior central gyrus. This fact is in entire agreement with our anatomical observations.

In dealing with the carnivora we are also in difficulties owing to the want of recent and exact observations on the topography of the excitable cortical centres for the movements of the trunk and limbs in their brains. It is evident, however, on comparing Hitzig's descriptions and the diagrams of his stimulation results with our figures that the area from which we trace the origin of the cortico-spinal tracts is practically co-extensive with the cortical centres for the limb and trunk movements discovered by Hitzig.

Hence, from our own stimulation experiments on the animals described in the present paper and elsewhere, and by reference to some of the chief classical excitation investigations of others, we must conclude that *the origin of the cortico-spinal tracts as delimited by our method is co-extensive with the excitable cortical area for the limbs and trunk.*

VII.

Finally the question comes: *Does the area of the origin of the pyramidal tracts coincide with any structural type of the cerebral cortex?*

There is a special interest in this question, as within the last few years the whole of the cerebral cortex of man, as well as of representatives of most of the mammalian classes, has been divided by several observers into a large number of areas, each characterized by its histological structure. The fullest communications on this subject we owe to Campbell [7], whose work was first presented to the Royal Society in 1903, and appeared in full in 1905, and to Brodmann [5], who has published a series of papers since 1903.

The importance of this sub-division of the cortex by histological methods lies in the fact that from difference in structure we may infer some difference in function, and a structural sub-division thus becomes parallel to a functional. This significance has been long recognized, indeed, in the early days of cerebral localization Bevan Lewis and Clarke discussed the relationship of definite areas characterized by the presence of "ganglionic cells" to the motor area as defined by Ferrier. Many other authors have applied the term "motor area" to the cortex of a certain region, or an area with certain structural peculiarities, only because some experiments had shown that movements could be obtained by excitation of it; and some, indeed, have been entirely dominated in anatomical investigations by more or less untrustworthy physiological data. Cajal [6], for instance, has included both central gyri in the "motor area," though recognizing the great difference there is between the structure of the cortex of each. The most noteworthy attempt to correlate function with structure has been made by Campbell, who has assigned specific functions to most of the cortical structural areas he has described. Brodmann has also recognized that it is a logical postulate that each area of specific histological structure has a specific function, but he, happily, has not burdened his observations with vague hypotheses regarding function. For it must be evident that the architecture of an area alone cannot be a trustworthy guide to its function; and that a physiological area should fall within or include a portion of a structural field is evidently no argument that this function is specifically related to that structural area, unless it can be definitely shown to be accurately co-extensive with it.

Excepting, perhaps, the calcarine type, with which the painstaking observations of Bolton have definitely associated the visuo-sensory function, the function of no structural area of the cerebral cortex has been made so probable as that which covers part of the anterior central gyrus in the primates. Campbell originally showed, in the brains of the apes that had been used by Grünbaum and Sherrington, that this structural area coincided very closely to that which responded to electrical stimulation. Vogt, on the other hand, has investigated the relation of the excitable cortex to this structural area by numerous stimulation experiments in the monkeys and other animals, and found that though in the anterior central gyrus the excitable area lies within Brodmann's area giganto-pyramidalis the two areas are not exactly co-extensive. The centres in the frontal lobes from which movements of the eyes can be obtained, and the focus from which mastication movements may be produced, correspond to other structural areas.

But no more definite proof of the physiological value of the histological sub-division of the cerebral cortex could be brought than the demonstration by anatomical methods that the area giganto-pyramidalis is exactly co-extensive with the field of origin of the upper motor tracts. Our previous conclusions have practically done this as far as man, the ape and monkey are concerned, for on the one hand we have found that it is only from the giant-cells of the anterior central gyrus and the corresponding portion of the paracentral lobule that the cortico-spinal fibres spring, while on the other these same giant-cells determine the extent of a structural field, the precentral area of Campbell, or the area giganto-pyramidalis of Brodmann. This area, it is true, is more extensive than that from which we have shown that the cortico-spinal fibres take origin, but there can be little doubt that from the portion of it remaining over the analogous cortico-bulbar fibres arise.

With this reservation we may then conclude that the origin of the pyramidal tracts in man, the anthropoid apes, and the lower monkeys is co-extensive with a "structural field."

In the lemur the area of origin of the pyramidal tracts is approximately the same as the histological field delimited by Page May and Elliot Smith [27] and by Brodmann [5], but it differs considerably from the "motor area" mapped out by Mott and Miss Kelley [22], who have placed the greater part of it behind the sulcus X.

The region from which in the dog we have found that the cortico-spinal fibres take origin corresponds closely with part of the area delimited by Brodmann [5]; it has practically the same posterior limit, and includes very little of the anterior sigmoid gyrus. According to Campbell [7] and Köppen and Löwenstein [16], the greater portion of the anterior sigmoid gyrus is included in this area, but with this statement we can certainly not agree. On the other hand, we found no changes lateral to the sulcus coronalis, or in the anterior portion of the gyrus sigmoideus, though these regions contained intact giant-cells. But if, as appears probable from stimulation experiments, it is from these unaltered regions that the cortico-bulbar fibres arise we may conclude that in the dog, too, the origin of the cortico-spinal fibres is co-extensive with part of a "structural field."

In the cat the area we have mapped out as the origin of the pyramidal tracts corresponds closely with the structural fields of Campbell and Brodmann.

CONCLUSIONS.

After division of the motor pyramidal fibres in the upper cervical region of the spinal cord there appears reactionary chromatolysis and atrophy of the cells in the cerebral cortex from which these fibres take origin. By operating thus on a series of animals (cat, dog, lemur, monkey and chimpanzee), and by utilizing two suitable cases in man, it has been possible to attain by histological methods the following conclusions:—

(1) The exact area of the cerebral cortex from which the cortico-spinal fibres subserving the limbs and trunk take origin is that shown in our figures and described in our text. In the primates this area lies entirely in front of the central (Rolandic) fissure.

(2) These cortico-spinal fibres arise only from the giant pyramidal cells of the infra-granular layer of this portion of the cortex, and these cells probably give origin only to cortico-spinal fibres.

(3) The area of origin of these cortico-spinal fibres coincides closely with the "excitable motor cortex" subserving the limbs and trunk, as this has been mapped out by recent investigations.

(4) This area of origin of the cortico-spinal fibres is co-extensive with part of a structurally differentiated cortical field.

REFERENCES.

[1] ACQUISTO AND PUSATERI. *Riv. di patol. nervosa e mentale*, 1897, vol. xviii.
[2] BALLET ET FAURE. *Rev. Neurolog.*, 1899, t. vii., p. 226.
[3] BEVAN-LEWIS AND CLARKE. *Proc. Royal Society*, 1878, vol. xxvii., p. 38.
[4] BOLTON, J. S. *Archives of Neurology*, 1903, vol. ii., p. 55.
[5] BRODMANN, K. "Beiträge zur histologischen Localisation der Grosshirnrinde," Mitteilungen i. to vii., *Journ. f. Psychol. und Neurolog.*, Bd. ii. to x.
[6] CAJAL. "Studien uber die Hirnrinde des Menschen," H. ii., Die Bewegungsrinde, Leipsig, 1900.
[7] CAMPBELL, A. W. "Histological Studies in Cerebral Localization," Cambridge, 1905.
[8] DEJERINE. "Anatomie des Centres nerveux," 1901, t. ii.
[9] DOTTO AND PUSATERI. *Riv. di patol. nervosa e mentale*, 1897, vol. xviii.
[10] EDINGER, L. "Vorlesungen uber den Bau der nervosen Zentralorgane," 7te Aufl., 1904.
[11] VAN GEHUCHTEN. "Anatomie du Système nerveux," 1900.
[12] GENTÈS ET BELLOT. *Compt. Rend. de la Soc. de Biologie*, 1905, p. 153.

[13] Grunbaum and Sherrington. *Proc. Royal Society*, 1901 and 1903, vols. lxvii. and lxxii.

[14] *Idem.* *Lancet*, 1901, December 21.

[15] Hoche. "Handb. der patholog. Anatomie des Nervensystems," 1904, p. 710.

[16] Koppen und Lowenstein. *Monatschr. f. Psychiatr. und Neurolog.*, 1906, Bd. xviii., p. 480.

[17] Marinesco. *Rev. Neurolog.*, 1899, t. vii., p. 358.

[18] *Idem.* *Deutsche med. Wochenschr.*, 1900, S. 351.

[19] Mellus. *Proc. Royal Society*, 1895, vol. lviii., p. 206, and *Journ. Nerv. and Ment. Diseases*, 1899, vol. xxvi., p. 197.

[20] von Monakow. "Gehirnpathologie," 2te Aufl., 1905.

[21] Mott and Halliburton. *Proc. Royal Society*, 1908, vol. lxxx., p. 136.

[22] Mott and Kelley. *Proc. Royal Society*, 1908, vol. lxxx., p. 488.

[23] Obersteiner. "Die nervose Centralorgane," 4te Aufl.

[24] Rothmann. *Deutsche med. Wochenschr.*, 1899, July 6.

[25] Schafer, E. A. "Quain's Anatomy," 1908, 11th ed., vol. iii., pt. i.
Idem. "Essentials of Histology," 1907, p. 426.

[26] Sherrington. "The Integrative Action of the Nervous System," p. 273, London, 1905.

[27] Elliot Smith and Page May. *British Assoc. Reports*, 1904, p. 760.

[28] Elliot Smith and W. H. Wilson. *British Assoc. Reports*, 1908.

[29] Vogt, O. *Verhandl. d. Anatom. Gesellsch.*, 1906, S. 74.

[30] Vogt, C. und O. "Zur Kenntnis der elektrisch erregbaren Hirnrindengebiete bei den Säugetieren," *Journ. f. Psychol. und Neurolog.*, 1907, Bd. viii., Erganzungsheft.

[31] Volsch. *Monatschr. f. Psychiatr. und Neurolog.*, 1906, Bd. xx., S. 470.

A NOTE UPON THE FARADIC STIMULATION OF THE POSTCENTRAL GYRUS IN CONSCIOUS PATIENTS.[1]

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ONE of the most important steps in our progress toward a more exact localization of function in the cerebral cortex was taken when, by a method of unipolar faradization, Sherrington and Grünbaum accurately delineated the confines of the so-called motor area. Their circumscription of this electrically excitable strip of cortex in the ape to the anterior wall of the fissura centralis (Rolandi) and to the adjoining surface of the precentral gyrus, has received confirmation through the more recent studies of myelinization, and through investigations into the finer histology of the cortex, which have shown that this area corresponds with the distribution of the Betz cells. Sherrington and Grünbaum's observations on the anthropoid brain, furthermore, have since been confirmed for man by Krause and Frazier; and I have had the occasion myself to stimulate the cortex of more than fifty anæsthetized patients with results in exact accord with those reported in 1901 from the Thompson Yates Laboratory of Physiology.

This surprising delimitation of what had previously been considered a wide territory to a relatively narrow precentral strip, left, without definitely proven function, the large portion of Munk's sensori-motor field—the old "motor area" of others—lying posterior to the central fissure. Conjectures have naturally arisen in regard to the function of this field—one from which motion may be elicited by undue strength of current—and the presumption that it is a sensory end-station has received support from certain histological studies (notably those of Alfred W. Campbell) as well as from a number of carefully observed clinical cases.[2]

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[1] Presented at a meeting of the American Physiological Society, December 30, 1908.

[2] It would thus appear, as is so often the case with disputed points, that there is an element of truth on both sides; for the original "motor area," embracing the convolutions on each side of the central fissure, appears to be sensori-motor, though not in Munk's sense —namely, with coinciding or superimposed afferent and efferent stations.

There can be no doubt but that lesions of the gyrus precentralis can lead to motor disturbances without recognizable sensory loss; on the contrary, a lesion limited to the gyrus postcentralis will almost inevitably inhibit normal free movements. I have seen a number of

The experimental study of sensation in animals, being limited to the interpretation of reflex actions, is most difficult and unsatisfactory. Needless to say, it is a matter which requires on the part of the subject not only an analysis of his sensory impressions, but their transference by descriptive speech to the observer, who in turn must interpret this description; for at best it is difficult to find words precisely indicative of many forms of sensation.

Owing to the impossibility of this necessary communication the making of observations on the sensory cortex of unanæsthetized animals would be as futile as during a state of narcosis. This, however, is not the case with man.

A year ago I reported, with Dr. H. M. Thomas, a surgical experience[1] in which at a second-stage operation an osteoplastic flap, previously made, was re-elevated, the dura reflected, the cortex incised, and a subcortical cystic tumour removed from the postcentral field of an unanæsthetized patient. The procedure, with the exception of accidental dragging upon the dura, and the final reclosure of the scalp, not only occasioned no discomfort, but was attended on the part of the patient by a lively and helpful interest in the performance. He even informed us of a focal sensory attack which occurred early in the proceedings, evidently incited by the preliminary palpation of the exposed brain. At the first session of the operation, under anæsthesia, the motor cortex had been electrically delineated, and we greatly regretted that after safely concluding the second stage we had not taken the occasion to test with the faradic current the postcentral areas.[2]

instances of this. Thus, in a patient with a glioma of the postcentral gyrus, partial hemianæsthesia was accompanied by marked motor disturbances, but no degeneration of the pyramidal tract was demonstrable at autopsy; and, further, an operative case with prompt restoration of normal motility after the evacuation of a postcentral, subcortical clot in a patient presumed to be hemiplegic, has been detailed in my Carpenter Lecture for 1906 (*New York Medical Journal*, January 26, 1907).

On a few occasions, in cases of focal epilepsy, characterized by a definite sensory aura in face or hand, I have extirpated a segment of the postcentral convolution corresponding with the level of the precentral strip, from which movement in the part concerned was elicited by cortical stimulation at the time of operation. Occasionally, though not always, a certain degree of hyperæsthesia has subsequently been demonstrable in the expected field; but this has always been a transient affair, clearing up in large part rapidly just as does a motor palsy resulting from the extirpation of a small segment of the precentral gyrus.

These observations have never been such as to justify definite physiological conclusions, for the extirpated segment has usually been small—a cubic centimetre of tissue possibly—and the deeper cortex lining the wall of the fissure must often have remained intact.

[1] "Removal of a Subcortical Cystic Tumour at a Second-stage Operation without Anæsthesia," *Journal of the American Medical Association*, 1908, vol. i., p. 847.

[2] A footnote describing a single, and the first experience in this direction, accompanied the article referred to above. In making a subtemporal defect for purposes of decompression in a case of brain tumour—a procedure usually concluded under anæsthesia at one sitting—it was necessary, owing to some meningeal bleeding, not easily checked, to postpone the opening of the dura to a second session. This was accomplished without an anæsthetic, and the exposed temporal lobe was stimulated, giving the patient certain definite auditory impressions which he clearly distinguished from the "buzz" of the vibrator.

In the past year, however, the exigencies of two surgical cases have given us the looked-for opportunity of making further observations upon the sensory field. It is the purpose of this paper to record solely the physiological aspects of these observations, although their surgical aspects are possibly of even greater importance; for if it will be possible in the future to pick out with an electrode areas of the brain from which the sensory aura of a focal convulsion has originated, we shall have advanced a long way toward the possible operative localization of subcortical irritative lesions of the immediately postcentral field.

Case 1. *History.*—The patient, a sturdy and intelligent boy of 15 years of age, was referred by Dr. H. M. Thomas to the surgical service of the Johns Hopkins Hospital on June 16, 1908, suffering from "convulsive attacks originating in right hand and right side of face." At the time of onset, two years before admission, these seizures were limited to a subjective sensory disturbance, characterized at first by periods of numbness in the little finger of the right hand, and finally by what was described as a "funny feeling" in the arm—a "gone sensation."

These purely sensory attacks increased in frequency and severity, and finally were followed by a twitching of the little finger; then by contractions involving the whole arm, and more recently by an extension of the motor disturbances to the right side of the face.

Though he had never himself observed an involvement of the right leg, the more severe seizures which occurred after his admission to the hospital were accompanied by a definite convulsive movement in the lower extremity as well.

A typical attack, many of which were observed, was preceded for a period of possibly ten minutes by a feeling of weakness or "goneness" in the arm. This was followed by tingling, roughly confined to the ulnar cutaneous distribution of the hand; then the arm would suddenly flex and be drawn up above the head. At this stage of the seizure he could occasionally check further progress of the "march" by grasping the affected arm with the left hand, and by pushing it violently to his side. No loss of consciousness accompanied the attacks, and usually, though not always, he could be made to speak during them. They were attended by a sensation of apprehension, but never by one of pain.

In the most severe seizures he would be drawn to the floor by an arching of the body to the right, with convulsive movements of the right side; but even in these attacks he was never known completely to lose consciousness. A distinct though transient weakness in the arm with irregular ataxic movements usually followed the attacks.

Beyond the presence of an incipient choked disc (hyperæmic stage), and a slight disturbance of pain and temperature sense over the postaxial surface of the hand and forearm, the physical examination was practically negative.

With the presumption that some organic lesion, possibly a tumour of the postcentral territory, would be found, an exploration was advised.

Operation (*first stage, June* 16, 1908.)—An area of the left hemisphere, as shown in the accompanying sketch (fig. 1), was exposed by the usual osteoplastic craniotomy. The hemisphere was found under slightly increased tension, but no abnormality was apparent.

Owing to the characteristic configuration of the central fissure, the landmarks of the exposed part of the hemisphere were perfectly clear, but the situation of the precentral gyrus was further assured by unipolar stimulation which elicited the usual sharp movements in wrist, in elbow, in shoulder and in face. The only subcortical investigation at this time was limited to the exploratory puncture of the postcentral gyrus in the vain hope that a cyst might thus be encountered. The wound was then reclosed.

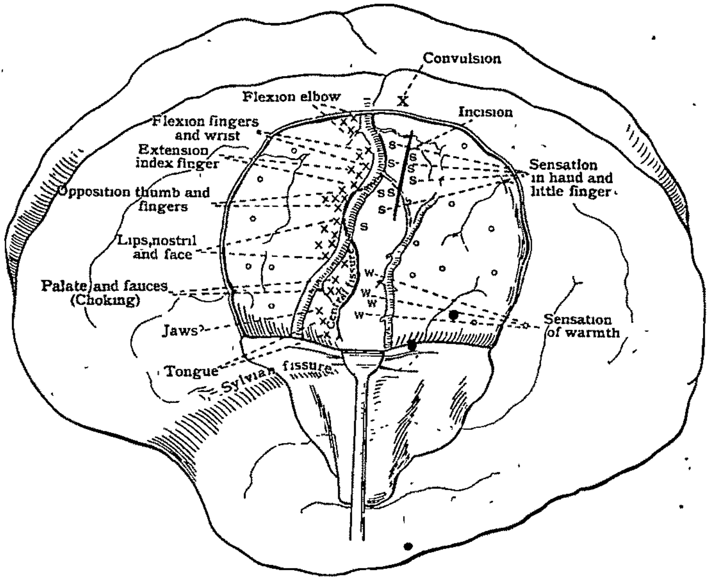

Fig. 1.

The boy's distressing seizures continued unabated, until finally it was determined that some more radical attempt should be made to disclose, if possible, their source of origin. It was hoped that if the cortex could be stimulated while the patient was conscious; we might thus obtain some evidence of the situation of the presumed lesion sufficient to justify a more extensive subcortical exploration.

Operation (second stage, July 6, 1908).—After a preliminary injection of $\frac{1}{6}$ gr. of morphia, and under primary anæsthesia by chloroform, the edges of the former incision were newly separated and the original bone-flap quickly re-elevated. The patient promptly regained complete consciousness, and while in this state, without occasioning discomfort, the dura was divided in the line of the previous closure, and the membrane was again reflected.

Cortical stimulation.—The following electrical observations were then made. The same movements as before were easily elicited with a mild faradic current from the precentral strip. These motor responses in detail and in the order in which they were obtained from the areas shown in the accompanying sketch (fig. 1) consisted of:—

(1) An opposing movement of thumb and fingers from points opposite to the unmistakable genu.

(2) Somewhat higher up, extension of the index finger.

(3) Still higher flexion of the fingers, which carried over to flexion of the wrist.

(4) At the upper margin of the exposed field, flexion of the elbow.

(5) Below the evident middle genu the movements, which required a somewhat stronger current, consisted of contraction of the side of the face.

(6) Movements of palate and fauces, a curious choking sensation being occasioned whenever they were obtained; and

(7) From the very lowest exposed part of the strip movements of the tongue occurred, and the patient experienced a sensation of movement in the lower jaw, though this was not certified by the observers.

It is to be noted that the subject was perfectly aware of these movements, and in the attempt to express the sense of feeling which was occasioned by them he said that it was the same sort of a drawing sensation appreciated when the muscles are "pulled" by peripheral stimulation—a familiar experience for him, owing to a long course of electrical treatment before his admission to the hospital. The movements, in other words, gave him the sensation of active muscular contraction alone.

Posterior to the central fissure, over an area roughly corresponding with the upper two-thirds of the exposed postcentral convolution (fig. 1), stimulation, with the same strength of current used to elicit the motor responses from the precentral gyrus, led to a sensation in the hand, arm and little finger—chiefly in the little finger—which was said to be exactly akin to the sensation that inaugurated his attacks. Stimulation lower down on the postcentral gyrus occasioned a sensation of warmth in the arm, but this was rather vague and indescribable.

It is to be noted that there were no painful sensations whatsoever, and further, that stimulation of the field in front of the motor strip and back of the postcentral convolution elicited no movements or sensations whatsoever, and the patient seemed to be unaware of the application of the electrode.

On elevating the upper edge of the dura at the point X (fig. 1) and placing the electrode there, an attack precisely similar to his usual attacks, inaugurated by the same sensory disturbances and followed by the customary abduction of the arm and arching of the body to the side, was produced. This lasted but a few moments, and was not followed by a general convulsion nor by loss of consciousness.[1]

In the light of these observations, a number of small vessels crossing the postcentral gyrus were doubly ligated and an exploratory excision 3·5 cm. in length was then made between the ligatures (fig. 1) and carried down to a depth of about 2·5 cm., unfortunately without disclosing any lesion. The procedure was practically bloodless, and the edges of the incised pia-arachnoid were subsequently approximated with three delicate split silk sutures. No sensation whatsoever accompanied the making of this deep exploration.

The dura was reclosed, the bone-flap replaced, and under a second primary chloroform anæsthesia the scalp was quickly resutured.[2]

Case 2. *History.*—An intelligent man, aged 44, began to suffer in August, 1907, with attacks of Jacksonian epilepsy of obscure origin. These attacks, beginning in and often limited to the right hand, were trifling at first, but soon became more numerous and more severe. They were usually preceded by an uncomfortable numb feeling in tilting of the head to the right. During the seizures, even of this degree of severity, though confused and unable to respond when questioned, he would not lose consciousness. On a few occasions, however—six in all—a severe general convulsion has occurred, in which the inaugural sensory disturbance has passed down the side of the body before consciousness was lost.[1]

His history otherwise and the physical examination were practically negative.

Operation (*first stage, December* 8, 1908).—The patient took his anæsthetic badly, and, owing to the attendant cyanosis, an unusual amount of blood was lost while elevating the large osteoplastic flap. It consequently was deemed best to postpone further measures to a second sitting, in the hope that with the patient unanæsthetized the dura might be opened and the suspected lesion sought for by cortical stimulation in case its situation was not immediately apparent.

Operation (*second stage, December* 14, 1908).—After the administration [illegible] anæsthesia from a few inhalations of

In order to be assured of the exact convolution from which these sensory responses occurred the gyrus lying anterior to the large vein was then touched about in the middle of the exposed field and opposite to what appeared to be a possible middle genu; a prompt flexion of the thumb into the palm of the hand followed this stimulus. The movement was unaccompanied by any sensation other than that of change of position of the thumb, which the patient promptly appreciated and described. In other words, there seemed to be no associated cutaneous sensation.

This latter observation was sufficient to certify the position of the fissura centralis (Rolandi), and although no further points on the precentral gyrus were subsequently stimulated, this same movement of the thumb was elicited as a control several times during the course of the later observations.

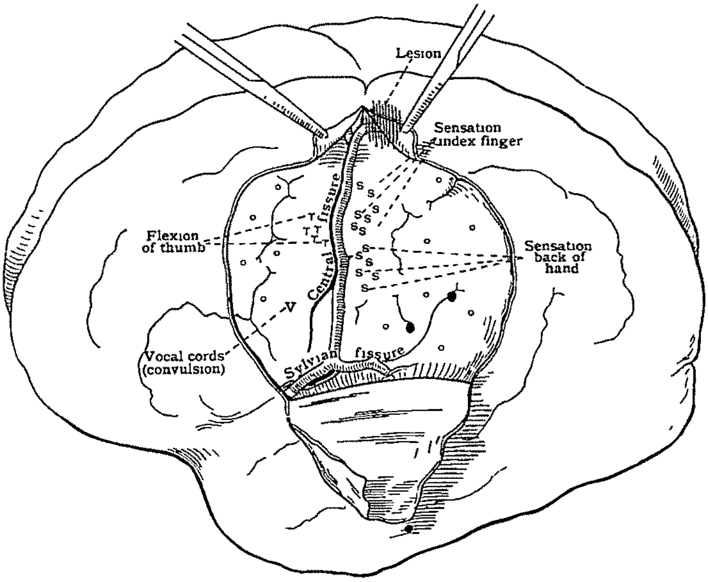

FIG 2.

The precentral and postcentral gyri having thus been identified, the outlying convolutions were "coursed" in order to see whether any subjective sensations occurred from stimulation of other than the postcentral gyrus from which the sensory impressions had first been obtained. Thus the crests of all of the exposed convolutions, at about the points marked by small circles in the sketch (fig. 2), were stimulated, with the elicitation of no sensory and, needless to say, of no motor responses whatever. Occasionally, in order to be sure that the current was sufficient for reaction, the primary stimulation of the area giving sensation of the index finger and of that giving movement of the thumb was repeated.

In coursing the exposed portion of the hemisphere as described, the operator finally touched, the point V, which immediately led to a curious, long-drawn, hoarse inspiratory sound, evidently due to closure of the glottis; this was followed by a general convulsion, the first observable evidence of which was a drawing-down of the right platysma and corner of the mouth, with depression of the chin and separation of the lips.[1]

Apprehensive lest a possible repetition of this unexpected convulsion might occur, and, from cyanosis and protrusion of the exposed cortex, occasion some damage, the operator desisted from further stimulation, although the exact area which occasioned the inaugural sensation of the patient's seizures had not been located.

At this juncture the administration of chloroform was resumed, and, under the anæsthetic, the upper and mesial edge of the postcentral convolution was more fully exposed by ronguering away a further portion of the skull at the upper edge of the cranial opening. This permitted an upward enlargement of the dural opening, as shown in fig. 2, and a lesion of the postcentral convolution, which was thought to be irremovable, was thus brought into view.

COMMENT.

These observations seemingly add confirmation to the view which ascribes a sensory function to the gyrus centralis posterior, but beyond this I shall make no endeavour to interpret their significance. Just what relation the afferent impulse bears to a motor response; just how stimulation of this supposedly sensory field is interpreted by the subject as sensory; why the motor response from stimulation of the gyrus centralis anterior occurs without any primary afferent impression, but only with the secondary consciousness of the movement—these and many other questions that arise are matters rather for the psychologist.

It is, perhaps, noteworthy that the sensations which were obtained were limited entirely to the hand and arm; and whether this means that there is an especially wide cortical representation for afferent impulses from this region, or whether these patients, owing to the characteristics of their seizures, possessed sensory centres from hand and arm which were unduly excitable, is difficult to tell, and will need further elucidation.

It is noticeable, in the last case, that the lesion which occasioned the seizures was found to lie higher on the postcentral gyrus than the areas which had been stimulated, so that presumably responses from the hand would have been brought about by stimulation there, just as they had been from a lower level of the convolution. It is possible, too, that the patient's attention in each case may have been so concentrated upon the affected hand that sensory impressions arising from leg, body and face passed unobserved. The fact that the sensations in the first case were precisely the same as those characterizing the aura of the patients' seizures would lend support to this view; but it was otherwise with the second case, for in this patient, though sensations occurred in the hand, they were other than those characterizing the inaugural stage of the fit and occurred over different skin fields.

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[1] It has seemed to me probable, from other experiences in the past, that the cortex in cases of epilepsy is more excitable than the cortex of non-epileptics; in the former it is traditionally easy, in fact, to occasion, even under anæsthesia, a Jacksonian fit which may run into a general convulsion. No apprehension, however, of this possible consequence of stimulation was felt in this particular patient, as such a weak current was being used. The numerous, rapidly executed and repeated stimulations of the seemingly non-excitable areas of the hemisphere may possibly have had an accumulative effect, so that when again approaching the lower part of the precentral convolution (Horsley's vocal-cord centre) the response to the stimulus was much more pronounced than was anticipated or was desirable.

SUMMARY.

Two patients, afflicted with epileptic attacks inaugurated in each instance by a sensory aura in the right hand, offered unusual opportunities, as related, for cortical stimulation while in a conscious state during a "second-stage" operation.

In both of them the situation of the central fissure was determined by obtaining characteristic motor responses from the precentral gyrus, these motor responses being attended by no sensation other than that of the forced change of position which accompanies similar movements elicited by stimulation of a peripheral nerve.

On the other hand, in both of these patients stimulation of the postcentral convolution gave definite sensory impressions which were likened in one case to a sensation of numbness, and in the other to definite tactual impulses.

In both of the patients, furthermore, stimulation of the outlying convolutions gave no response whatsoever, either of a subjective sensation or of active movement.

ON CERTAIN CASES OF ACUTE TREMOR OCCURRING IN CHILDREN.

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HAVING no pathological evidence to bring forward as to the nature of these cases, they have been described in the title in strictly clinical terms, the phrase "acute tremor" being used for the sake of brevity and to enforce the analogy between this condition and that named by Leyden "acute ataxia" (now attributed to encephalitis cerebelli). This latter consideration is not unimportant; for I believe, for reasons to be given later, that the cases here detailed are examples of encephalitis of the mid-brain and its connections.

The tremor which, with its acute onset, is characteristic of these cases, corresponds exactly with that described by Gordon Holmes (2) as associated with destructive lesions occurring in the cerebello-rubro-spinal system. It is a slow, regular tremor which at its worst is continuous except during sleep, although increased by voluntary movement, and which when less severe shows itself only on voluntary movement, or during periods of excitement. It may be universal in the head, tongue, and muscles of the trunk, abdomen, and limbs, and it is sometimes more definite in the proximal than in the peripheral parts of the extremities. In some of these cases the appearance of the child is very like that of one shivering all over from cold. In rate, the tremor is about five oscillations per second. Combined with the tremor is hypertonus, giving to the limbs some degree of uselessness, and rendering their movement slow, stiff, awkward, and this in the absence of true spasticity. There is little or no loss of power. In addition, there may be other signs present from the involvement of the parts of the brain in the immediate neighbourhood of the cerebello-rubro-spinal system, or from the presence of other foci of disease elsewhere in the central nervous system.

The important points, therefore, in these cases are three in number, the acute onset, the tremor and the hypertonus, the last two differentiating the condition from cerebellar encephalitis in which ataxia and hypotonus are found, with the possible additions of nystagmus and scanning speech.

Where these cases have been reported, as by Batten [2], they have been grouped as cases of cerebellar encephalitis, but it would appear that they warrant separation from these, a view which I put forward in a paper concerning encephalitis [3], and which is also held by Poynton [4] in a communication in which two such cases are described, one of which is Case 3 of this series.

The cerebello-rubro-spinal system consists of two parts—the cerebello-rubral and the rubro-spinal. In the former the dentate nucleus of the cerebellum is connected with the red nucleus and optic thalamus of the opposite side by the superior cerebellar peduncle; in the latter the red nucleus is connected by the tractus rubro-spinalis (Monakow's bundle) with the opposite side of the spinal cord. In the pons Monakow's bundle passes close to the nucleus of origin of the seventh cranial nerve. In this system, therefore, are two decussations, each of which occurs in the immediate neighbourhood of the red nucleus, the superior cerebellar peduncles crossing before entering the nucleus, and Monakow's bundles crossing after leaving it. From this it follows that if the tremor be due to destruction of any part of the system, the movement will be on the same side as the lesion, unless the red nucleus itself or its immediate surroundings be involved, in which case it will be on the opposite side.

It will be convenient now to give an account of the clinical histories of the cases, but before doing so I must express my indebtedness to Dr. Voelcker, Dr. Colman, and Dr. Poynton for permission to use the patients I watched when Medical Registrar to the Hospital for Sick Children, Great Ormond Street. My thanks are also due to the Casualty Medical Officer of that hospital, Dr. L. G. Parsons, for his kindness in allowing me to see the last patient here reported, which was brought up to his department.

CASE 1.

J. A. P., a boy, aged 2½.

History of illness.—In the first week of October, 1906, the child, when seated at table, was noticed to go suddenly "all of a heap," with his mouth drawn to the left. He was unconscious, and remained so for three weeks, although he was very irritable when disturbed. His limbs were observed to tremble. After three weeks he regained consciousness and was found to have lost his speech. He lay helpless in bed, with the shaking movements continuing all the time except during sleep. He was admitted to the Hospital for Sick Children, under Dr. Colman, on November 12, 1906.

Past history.—Had always been a very healthy child, and had learned to walk and talk well.

Family history.—The patient was the elder of two children; the other is well. The mother had no miscarriages and there was nothing of importance in the family history.

On admission (November 12).—The child was very well nourished, showing only slight bony signs of rickets. Apart from the nervous system no abnormalities were present.

Mentally the child was very irritable, although probably not in pain, and was extremely emotional. He cried very loudly, and would change from this into laughter, which was excessive and hysterical, resembling that of a child being tickled. He made no intelligible efforts to speak, but appreciated noises and things to eat. No diminution of emotional expression could be detected.

Tremor was seen in the head, tongue, trunk, and all four limbs. It was present except during sleep, but it was increased on voluntary movement. In the limbs it was more marked in the proximal than in the distal parts. In the hands it was a slow rhythmic tremor of about five per second. The whole appearance of the child was like one shivering from cold. In addition the voluntary movement of the limbs was performed slowly and stiffly as in a case of paralysis agitans. On the right side there was possibly some spastic rigidity.

Examination of the cranial nerves showed that there was some affection of the sixth and seventh nerves on the right side. The others appeared normal. The optic discs and the tympanic membranes were normal.

The reflexes showed little change. The right abdominal reflexes were absent, the left being just obtainable. The pupil, corneal and palate reflexes were normal. The tendon reflexes were brisk and equal on the two sides, and the plantar responses flexor. The jaw-jerk was active. There was reflex incontinence of urine.

Course of disease.—The child was seen at intervals for eighteen months. During the first weeks the evening temperature was slightly raised, as is seen in recent cases of acute poliomyelitis. A month after admission the cranial nerves and the abdominal reflexes had become normal and the tremor less marked, so that it was present only on voluntary movement. The hypertonus was still evident. A month later he was able to walk a little.

During the rest of the time slight further improvement took place. By March, 1908, all signs of the tremor had disappeared, but the movements of the limbs and of the head were slow and stiff. He was able to walk very fairly well by this time. Mentally he had made practically no improvement at all. He was a little quieter, but quite idiotic. He still laughed and cried hysterically, was extremely greedy, dirty in his habits, and made no intelligible effort to talk.

In February, 1908, he had three generalized convulsions which were not followed by any observable deterioration. In May, eighteen months after the onset of the illness, he had a severe fit which proved fatal No *post-mortem* examination was possible.

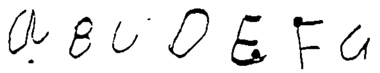
Localization.—From the affection of the sixth and seventh nerves on the right side, the general tremor and great emotionalism, we may surmise that the lesion was an extensive one involving the mid-brain and pons. But in addition it may well be that the cerebral cortex sustained some damage, which view may be supported by the prolonged unconsciousness at the time of the onset, the profound mental change, and the later development of epilepsy.

(This patient was shown by the writer as a case of encephalitis, chiefly affecting the mid-brain, at the Clinical Society, April, 1907.)

CASE 2.

L. S., a girl, aged 5¼.

History of illness.—On August 3, 1907 she complained of pains in the back and limbs and could not put the heel of the left foot to the ground On the next day "shivering" of the right hand was noticed and she was unable to walk. She had no apparent feverishness, no convulsions, but talked in her sleep at night and seemed drowsy. She was kept in bed for a week. Only slight improvement was made until she was admitted to the Hospital for Sick Children on August 20, under the care of Dr. Colman.



A Specimen of the Handwriting of L. S., aged 6½ years, 16 months after onset of illness (Case 2).

Past history.—Had always been a big, strong child. Had measles five months ago and whooping-cough previously.

Family history.—Only child; the mother had had no miscarriages. Both paternal grandparents died of consumption. Maternal grandmother and uncle died insane at the ages of 62 and 29 years respectively. Parents healthy

On admission (August 20, 1907).—The child was well developed and nourished. She was irritable and cried when disturbed; her memory and speech were good. When sitting up the head tended to assume the cerebellar attitude, the right ear approximating to the right shoulder and the face turning to the left with the chin raised. There was slight lateral nystagmus to right and left, none vertically. The gait was very ataxic and the child could not walk alone, falling backwards. The left limbs showed ataxic movements in both arm and leg. On the right side there was no ataxia, but tremor and hypertonus, the tremor being of the intention type and the voluntary movements of the limbs being slow and stiff. The cranial nerves were normal and the deep reflexes brisk, but rather less so in the left than in the right limbs. There was no evidence of involvement of either pyramidal tract.

Course of illness.—Very rapid improvement took place. The child quickly became docile and quiet. Within a week the ataxy and the tremor diminished considerably, and within a fortnight she could walk unaided. A month later she could walk and run well, but some little ataxia showed itself in the left leg when she tried to kick a ball on the ground with the left foot. By this time the tremor had quite gone from the right leg, but was still just recognizable in the right hand.

In December, 1908, sixteen months after the onset of the illness, the child still showed a very slight tremor in the right hand when trying to do any fine action with her arm unsupported, but she was able to feed herself perfectly well. A specimen of her handwriting at this date shows how very slight was the tremor with her arm supported. The movement is at its worst when she is wakened from sleep, or when she is nervous. Mentally she was perfectly normal, and she appeared very strong and healthy in every other respect.

Localization.—From the presence of the ataxia on the left side, the cerebellar tilt of the head and the slight nystagmus, it may be supposed that the lesion was in the cerebellum, and that the tremor on the right side was due to the involvement of the right dentate nucleus.

CASE 3.

A. C., a girl, aged 13 *months.*

History of illness.—The mother stated that the child had been seized with convulsions five weeks previously (*i.e.*, the beginning of August, 1908), and that these had continued occurring several times a day. After four weeks the child seemed slightly better for two days, but then relapsed, being convulsed and unconscious. Something wrong with the eye had been noticed for the last month. There had been no gastro-intestinal disturbance.

The mother was an Italian and could speak but little English, and I think that the history given may be correctly interpreted thus: that the child was seized suddenly with an illness in which movements of the limbs were present, and that for the first week the child was unconscious or very drowsy, and as this condition passed off the squint was noticed for the first time. This view of the history is supported by the fact that the child when first seen was neither convulsed nor unconscious, as the mother had stated. She was admitted to Dr. Voelcker's ward at the Hospital for Sick Children, under the care of Dr. Poynton.

Past and family history.—Nothing was noted on these points except that the parents had had no fits.

On admission (September 7, 1908).—The patient was a very fat child, showing rickety changes of the head and chest. She was conscious and was not irritable. The pupils were small, reacted poorly to light, especially on the right side, but dilated well under atropine. There was complete paralysis of the right external rectus, and there was definite weakness of the right side of the face, the eye not closing completely on this side. There was some doubtful weakness of the left external rectus.

The tremor was constantly present, except during sleep, in the head, trunk and limbs, being most definite in the lower extremities and least definite in the head. It was increased by voluntary movement and was in every other way similar to the rhythmic movements described in the other cases. It was very difficult to say that there was any definite hypertonus of the limbs. There was no loss of motor power. The reflexes were unaltered; the abdominal reflexes were all present, the knee-jerks brisk and equal, and there was no ankle-clonus. On attempting to elicit the plantar responses, voluntary movements of flexion were obtained. Lumbar puncture was performed on September 9, and clear fluid was obtained, which was sterile and showed no deposit or clot, and in which no cells or organisms were found.

Course of illness.—During the next week there was a good deal of improvement, the tremor becoming less and showing only on voluntary movement.

Localization.—From the affection of the sixth and seventh cranial nerves on the right side, the lesion, or part of it, may be thought to be pontine.

CASE 4.

L. K., a girl, aged 2¾.

History of illness.—On October 24, 1908, the child suddenly became very drowsy and passed rapidly into an unconscious condition. There was no convulsion. She remained unconscious for four days. The tongue was noticed to tremble from the first day of the disease, but no tremor was seen in the limbs until the child sat up towards the end of the first week, when it was observed in the head and left limbs when voluntary action was undertaken. The movement was described as giving an appearance of "shivering." The squint and facial weakness were not noted. Rapid improvement had occurred. She had become greedy and was very noisy at night. During the last week she has begun to try to talk again.

Past history.—Physically well, but was described as being backward in her talking and always passionate and "spoilt." Habits not clean. Was said to have had convulsions at birth and at 6 weeks of age; no fits since then.

November 10, 1908.—Was seen as an out-patient at Paddington Green Children's Hospital seventeen days after the onset of the illness. She was found to be a strong, well-developed child. She was extremely passionate and irritable, and cried very loudly under examination; but there was no hysterical laughter, as in Case 1. There was weakness of the left external rectus and of the left facial muscles, the eyelids not being well closed on this side. Slight tremor was seen in the tongue, in the left arm and leg. In the limbs it was only present during volitional movement. Owing to the tremor in the legs she could not stand, but there was no actual loss of motor power in the limbs. The voluntary movements of the affected limbs were slowly and stiffly performed. So far as could be made out, the reflexes were unaltered; it was impossible to test the abdominal reflexes owing to the child's behaviour.

Course of illness.—When seen a fortnight later she was much more docile.

The cranial nerves and the left arm were normal, but the left leg showed slight tremor still, although she was beginning to walk alone. The abdominal reflexes were normal.

Within six weeks of the onset of the disease the child appeared to be perfectly well, walking, sleeping, and behaving well. She was said to be using more words than before her illness.

Localization.—It is probable that in this case there was a small lesion involving the sixth and seventh cranial nerves and the rubro-spinal (Monakow's) tract in the left side of the pons.

Case 5.

W. W., a boy, aged 1¾.

History of illness.—At the end of July, 1908, he suddenly began to scream violently, and appeared frightened, holding on to a chair. He was pacified after a few minutes, and as he got very drowsy he was put to bed. The next day continuous trembling was noticed in all the limbs. From this time he was unable to walk, he lost the few words he had learned, and became very noisy, nervous, and greedy. The trembling continued, but became gradually less.

Past history.—Was a strong child, good tempered, able to walk, and was beginning to talk before his illness. No other diseases.

Family history.—The child's mother had always been very nervous and "hysterical," and had lately developed epilepsy. She had had no miscarriages. Father strong. One other child, aged 8, well.

November 23, 1908.—When first seen, four months after the onset of the disease, he was found to be a strongly-built child, and very fat. He was very noisy, crying and laughing hysterically. He could not talk at all, and was mentally deficient and greedy. The tremor was seen in the limbs, showing only on voluntary movement; in the legs it was sufficient to prevent his standing. The movement of the limbs was slow and stiff in character. There was some jerky movement of the head and eyes, but no tremor or nystagmus. The cranial nerves and the reflexes were normal.

Course of illness.—A fortnight later he was a little quieter in his behaviour; the tremor was less, and the child was beginning to stand with assistance. There was no mental improvement. There had been no fits.

Localization.—Part of the lesion may be supposed to be in the mid-brain, involving the red nuclei.

Case 6.

A. Q., a boy, aged 15 *months.*

History of illness.—On October 19, 1908, the boy was put to bed well and, although he slept with his mother, nothing was noticed until the morning, when he was found to be very drowsy and to be "shivering" all over, but the limbs were not paralysed. No fits were seen, nor was the boy unconscious. The movements very rapidly got better, but the child was not able to walk for about a fortnight on account of the trembling of the legs. A week after the onset of the illness the right arm became useless, and hung down by the side. This paralysis was not accompanied by any constitutional symptoms, and was first noticed one morning after the child had had a normal night's rest.

Past history.—Had always been a fairly strong child, but was subject to attacks of vomiting. Had no previous illnesses except these bilious attacks.

Family history.—Only child. No neuropathic inheritance could be traced. No cases of infantile paralysis were known of in the neighbourhood.

January 29, 1909.—When first seen he was found to be a strong, fat boy, showing no trace of the tremor described by the mother. He was able to walk well, and could say more words than before the illness. No mental impairment could be found. The right arm was paralysed, and hung flaccid at the shoulder. There was slight power in the fingers of the right hand. According to the mother's statement, practically no improvement had taken place in the condition of this arm.

Localization.—It would appear that a slight lesion had occurred somewhere in the cerebello-rubro-spinal system, and that there had been a relapse a week later (the usual time for a relapse when such does occur), affecting the cervical enlargement of the cord.

ETIOLOGY.

It is necessary here to submit the clinical evidence which supports the contention that we are dealing with cases of encephalitis, using that term as indicating a disease of the brain similar to that which is known as acute poliomyelitis when it affects the spinal cord. In Case 6 we have a spinal lesion of the acute poliomyelitis type, occurring as a relapse in a child who had developed tremor a week previously. But apart from such evidence as this, acute poliomyelitis has certain well-marked characteristics which should be reproduced in polio-encephalitis with some modification, and these points may now be considered.

(*a*) The child who is attacked by poliomyelitis is very frequently the strong and fat, the delicate and thin children being notably spared. This rule held good in the six cases here reported, as it has in all other cases of encephalitis that I have seen. (*b*) The seasonal incidence of these cases corresponds to that of acute poliomyelitis, one being in July, two in August, and three in October. (*c*) The age-incidence, from 13 months to 5½ years, corresponds exactly to that at which the spinal disease is commonest. (*d*) The amount of constitutional disturbance at the onset of the disease is very variable, as in poliomyelitis, although it is natural to suppose that convulsions and unconsciousness are likely to be of more common occurrence. In Case 1 the child was taken ill with a convulsion and was said to have been unconscious for the following three weeks, while in Case 2 there was no fit and only slight drowsiness was observed. (*e*) In poliomyelitis the onset is acute and the harm done reaches a maximum within a few hours; anything like weakness becoming gradually an absolute paralysis is not seen. So, too, in these cases the disease is acute and its effects complete and absolute; but here care must be taken in examining the clinical history of the illness, for the same difficulty is experienced as has been found in dealing with cases of cerebellar encephalitis. Should the child be drowsy or unconscious the tremor may not be seen or may be regarded as signifying a convulsion, so that one may hear that the trembling came on some days after the onset of the illness. This possibility is well instanced in Case 4, in which the mother stated that the child was unconscious for four days and when sat up was seen to have shaking of the limbs. With questioning, however, it transpired that the tongue was seen to tremble when the child was fed on the first day of the illness. (*f*) As in poliomyelitis, the tendency of the disease is towards improvement or total recovery, while relapses are very exceptional. (*g*) Examination of the cerebro-spinal fluid was negative in the one case in which it was undertaken (Case 3), as it is in spinal cases.

In none of the cases here reported had the disease been preceded by one of the acute specific fevers of childhood within three months of the date of onset, a point which has been noted before in dealing with encephalitis and which by some has been used as an argument against the condition being the same disease as poliomyelitis. In my experience such a sequence in true cases of acute poliomyelitis is by no means so common as some have seemed to imply.[1]

It may be pointed out, without comment, that in the five cases in which it was possible to get a true family history two showed a strong neuropathic taint, while the third child was described as spoilt and passionate and suffered from nocturnal enuresis. I do not think that any such association has been noted in cases of acute poliomyelitis.

SYMPTOMATOLOGY.

The acute onset, the tremor and hypertonus are the important characteristics of this group of cases, and have been sufficiently described above.

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[1] In order to substantiate this point I have examined the records of the Hospital for Sick Children for cases of acute poliomyelitis whose onset occurred in 1906. Of these I found fifteen, and in only one was there a history of an acute specific fever within three months of the paralysis, while eight had never had any of the exanthemata at any time. The monthly incidence of these cases was August 5, September 6, October 3, November 1.

In addition, there may be other signs due to involvement of some part of the nervous system other than the cerebello-rubro-spinal tracts. Thus the anterior horn-cells of the spinal cord may be affected (Case 6), or the cerebellar involvement may produce ataxia (Case 2), or the sixth and seventh cranial nerves in the pons may be damaged (Cases 1, 3, and 4). Excessive emotionalism was present in Case 1, and to a less extent in Cases 4 and 5, and may perhaps be regarded as being produced by damage of the optic thalamus. Mental deterioration (Cases 1 and 5) appears to be a serious symptom, and if present may perhaps be thought to be due to an associated polio-encephalitis superior, involving the frontal areas of the cerebrum as has been described by Batten [1]. Hemiplegia may occur, with the tremor displayed on the paralysed side, or a third nerve paralysis.

DIAGNOSIS.

This naturally falls under two headings: the character of the lesion and its localization.

It is not maintained that all cases of acute tremor are to be regarded as due to polio-encephalitis, but only that certain of them may be placed in this group. The diagnosis can to a large extent be made by the exclusion of other possibilities, but those etiological factors already mentioned—the type and age of child, the season of the year, the character of the onset, and the course of the disease—may be of use in settling the matter. The cerebro-spinal fluid shows no abnormalities. The most likely causes, apart from polio-encephalitis, are a tuberculous tumour or a thrombosis due to syphilitic endarteritis.

Of the former I have seen one example, the tremor being seen during the first few days in a case of tuberculous meningitis, and being due to a small mass found after death in the dentate body of the cerebellum. In such a case the child, although it may recover, is generally far from well nourished and robust; there may be signs of tuberculosis elsewhere, optic neuritis may be present, and the cerebro-spinal fluid will show an excess of lymphocytes. I have seen no case in which the tremor could be said to be due to a syphilitic thrombosis; but judging by what occurs where other parts of the brain are affected, the diagnosis should be clear by the presence of signs of inherited syphilis (particularly in the eye-grounds), and the possible warning convulsions or "fainting attacks." Here, again, lymphocytes would be present in the cerebro-spinal fluid.

The localization of the lesion has been discussed. The tremor and hypertonus distinguish these cases from the true cerebellar ones which show ataxia and hypotonus. The part of the cerebello-rubro-spinal system attacked is diagnosed by the accompanying signs if such are present.

PROGNOSIS.

There is a general tendency towards improvement. In all six cases the tremor rapidly diminished, and in a few weeks or months became inappreciable. Similarly the cranial nerve paralyses disappeared. Of far more serious import is the mental deficiency, which may remain after the acute attack is over, and which, where it was present in these cases, remained unimproved. In one of these epilepsy developed and proved fatal.

REFERENCES.

[1] BATTEN. "Cerebral Symptoms due to Encephalitis," *Med. Soc. Trans.*, vol. xxvii., p. 116.
[2] HOLMES. "On Certain Tremors in Organic Cerebral Lesions," *Brain*, 1904, p. 327.
[3] MILLER. "On Acute Poliomyelitis and the Allied Conditions in the Brain," *St. Mary's Hosp. Gaz.*, December, 1907.
[4] POYNTON. "Some Unusual Sequelæ of Convulsive Seizures in Childhood," *Lancet*, October 31, 1908.

A CLINICAL STUDY OF OPTIC NEURITIS IN ITS RELATIONSHIP TO INTRACRANIAL TUMOURS.

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CHAPTER I.

Introductory.

THIS paper is based entirely on the clinical records of patients admitted to the National Hospital between the years 1900 and 1907. It includes the cases of cerebral tumour admitted to the hospital during those years in which the diagnosis was confirmed either by *post mortem* or by the operative removal of the tumour. The work of summarizing these records was suggested to me by Mr. Marcus Gunn, and I have to thank him and the staff of the National Hospital for the ready permission that they granted me to utilize their most valuable records. I have specially to thank Mr. Gunn for his constant kindly encouragement and advice during the somewhat tedious task of abstracting and summarizing about 400 cases in all. Much of the value of the paper depends on the fulness and accuracy of his notes of ophthalmoscopic observations made from week to week on his visits to the hospital. Many of these patients I saw along with Mr. Gunn and was able to watch from day to day; for the ophthalmoscopic notes in some cases admitted while he was on his holidays I alone am responsible.

The worth of any statistical paper must rest to an equal extent on the care taken in making the original observations and on the method in which the statistics are used. In certain portions only of this paper are the numbers sufficiently large to justify the use of percentage values. If, for the sake of completeness, I have given percentages in other places where the numbers are smaller, I have been careful at the same time to give the actual numbers. And I hope that, apart from any value the paper may have in itself, it will be of use to some future worker seeking to collect a very much larger number of cases from the literature of this subject.

The value of any conclusions I have formed will vary proportionately to the number of cases on which they are based. At best, any clinical statistical paper is of utility only in suggesting hypotheses on which to base experimental or histological research, and I hope that certain lines of work which this paper indicates may prove productive of more definite results, either in my own hands or in those of some other worker in the same field.

The classification of tumours as belonging to different regions of the brain has been a matter of some difficulty, and must be, to a certain extent, arbitrary. I have endeavoured, as far as possible, to classify each tumour in the region to which it most properly belongs. But tumours frequently stray outside the boundaries by which we separate different cerebral lobes from one another. The greatest difficulty in this respect has been with the class of tumours which are generally known as subcortical. Cortical tumours are not restricted to the cortex alone any more than deep or subcortical tumours remain confined to the underlying white matter of the brain. Many of these latter, on the one hand, invade the overlying cortex, or, on the other hand, extend to the basal ganglia, and in some cases it is a matter of difficulty to judge whether one is justified in dealing with them as subcortical tumours. On the other hand, there are one or two tumours classified as occupying the precentral and postcentral regions which in all probability had originated deep in the white matter, and involved the cortex secondarily, but to such a great extent that they had to be classified as cortical tumours. This topographical diffuseness of tumours involves a certain amount of looseness in classification, but I do not think it invalidates in any essential manner the arguments I have based on this series of cases.

It must be pointed out that certain factors, independent of the optic neuritis itself, have an important influence on clinical observations, such as are recorded in this paper. The most important of these factors is the duration of the patient's life after the appearance of the disc-changes. With certain tumours, *e.g.*, in the frontal lobe, the patient may live for months or years; while tumours of other regions, such as the fourth ventricle, very quickly terminate in death. Further, craniectomy has a very important influence on optic neuritis, and this operation is performed with varying frequency and at different periods according to the site of the tumour, *e.g.*, cerebellar tumours nearly always undergo operation, rolandic tumours are usually operated on early, while pontine tumours very seldom come into the surgeon's hands. These different conditions have been taken into consideration, as far as possible, in the conclusions stated in the paper.

The complaint may be made that in the following paper I pay little or no attention to the work of other writers on this subject. My reason for not doing so may be very briefly stated. This paper is entirely clinical, and the statements made in it are based on observations on a particular series of cases. The number of cases is not as large as might be desired, but they have been very carefully observed, and what I have sought to do is to form for myself from these cases as clear a clinical picture as I can of optic neuritis as it occurs in cerebral tumours. It is that picture which I desire to place before my readers, and, though critics may find fault with the perspective or the proportions, I would ask them to realize that in depicting it I have striven to keep myself as free as possible from the bias of preconceived notions or authoritative teaching.

CHAPTER II.

The Incidence of Optic Neuritis in Tumours affecting Different Regions of the Brain.

The 200 cases on which the paper is based fall into the following groups:—

Precentral	30	Extra-cerebellar	19
Postcentral	25	Pontine	15
Temporo-sphenoidal	13	Third Ventricle	4
Deep or Subcortical	32	Fourth Ventricle	5
Optic Thalamus and Mid-brain ...	15	Pituitary	2
Cerebellar	33	Multiple	7

Forty of these 200 cases (exactly 20 per cent.) showed no optic neuritis at any period in their history up to the time of death or operative cure. Of these forty cases, however, there were four in which a primary or pressure atrophy of both nerves occurred. The development of this condition was in each case due to the direct pressure of the tumour on the chiasma. In two the pituitary body was the site of the tumour; in the third the tumour grew from the floor of the third ventricle, and in the fourth case (No. 25) the tumour originated from the under-surface of the frontal lobes. In each of these cases the atrophy developed without any preceding or accompanying œdema of the nerve head, and in all the loss of vision was very marked and preceded the paling of the discs. This was specially notable in the case (No. 25) of frontal tumour.

In this connection another case of frontal tumour may be cited (No 1) in which the right lobe was invaded by a tumour which extended up from the orbital surface and also downwards and backwards, involving the tip of the right temporo-sphenoidal lobe, compressing the right optic nerve, the right side of the chiasma and the right optic tract. There was definite left hemianopia, but moderately good central vision in the left eye, though central vision was absent in the right eye. The right optic disc was normal in appearance, though there was a tinge of red in the upper part of the disc which might be taken to indicate commencing capillary congestion. The left optic disc showed intense optic neuritis with much greyish opacity stretching all round and over the neighbouring retina for some distance, one or two hæmorrhages on each side, and a swelling of two dioptres. In this case, then, the pressure of the tumour gave rise to blindness in the right eye, though no primary atrophy had, at the time of death, shown itself in the right disc. In another case which I have recently seen (not included in the present series) the conditions were somewhat similar; here, however, there was a marked primary atrophy in the left eye with an intense neuritis in the right. *Post-mortem* examination in this case revealed a large frontal tumour excavating the under surface of both frontal lobes, but more on the left side than on the right. The left nerve was directly pressed on by the tumour.

These cases show that direct pressure on the intracranial portion of the nerves or on the chiasma may cause loss of vision and pressure atrophy without causing optic neuritis. I shall have occasion to return to this subject later in the paper.

Table I. shows the regions of the brain most frequently affected by tumours without developing neuritis. Twenty of these cases come under the heading of subcortical and pontine tumours, while we see that all the cases of temporo-sphenoidal, cerebellar, and fourth ventricle

TABLE I.

Relative frequency of Cases without Neuritis.

	Cases	No Neuritis	Percentages with no Neuritis
1. Subcortical ...	32	14	43·75
2. Pontine ...	15	6	40
3. Extra-cerebellar ..	19	5	26·315
4. Optic Thalamus and Mid-brain ...	15	3	20
5. Precentral ...	30	5[1]	16·6
(*Excluding Case of Primary Atrophy*)	(29)	(4)	(13·793)
6. Multiple ...	7	1	—
7. Postcentral ...	25	3	12
8. Third Ventricle and Pituitary ..	6	3[2]	—
9. Temporo-sphenoidal ...	13	—	—
10. Cerebellar ...	33	—	—
11. Fourth Ventricle ...	5	—	—
Totals ...	200	40	—

[1] Including one case of primary atrophy.

[2] All cases of primary atrophy.

tumours develop neuritis. We must examine in some detail all the cases of subcortical and pontine tumour to see if we can find any means of differentiating between the cases which develop neuritis and those which do not. We must further examine in detail why, in certain cases, neuritis does not arise in connection with tumours of other regions which, as a rule, are associated with it.

Tumours of the pons afford us the simplest problem to start with. Here we find that the development of neuritis is due invariably to the tumour spreading beyond the limits of the pons to invade neighbouring structures. Taking these cases in detail, it is definitely stated in the *post-mortem* reports that the cerebellum was involved in seven of the cases, in five it was not involved, while this point is left doubtful in three cases. Of the seven cases where the cerebellum had been invaded by the tumour, five had, and two had not, neuritis. In these two the invasion of the cerebellum was only of the slightest degree, being restricted to the white matter of the middle peduncle (Cases 173 and 174), so that though, strictly speaking, they must be included amongst the cases in which the cerebellum is involved, practically they should be included with cases of non-invasion of the cerebellum. Of the five cases where it is definitely stated that the cerebellum was not involved, four had no neuritis and in the other (Case 268) there was slight blurring of the right disc before operation with no changes at all in the left eye, though subsequent to operation quite definite slight neuritis appeared in both eyes. *Post mortem*, this case revealed huge masses of lobulated tumour almost entirely concealing the pons and invading the mesencephalon right forward. In the three cases in which the notes do not make any statement as to the invasion of cerebellum neuritis was definite. There can be little doubt that in all three cases the cerebellum was affected, since it is stated in one (Case 175) that the tumour extended on the left side from the exit of the fifth nerve to the middle of the inferior olives, and at the junction of the pons and medulla involved nearly the whole transverse section of the bulb. In the other two the symptoms certainly indicated cerebellar invasion to such an extent that both were operated on for cerebellar tumour. The examination of these cases may lead us justly to the conclusion that tumours of the pons Varolii do not produce optic neuritis, except by secondary invasion of the adjoining regions, more especially the cerebellum; and this, I think, fully accounts for the well-known fact that pontine tumours only give rise to optic neuritis very late in their history. In other words, when the tumour has invaded these neighbouring structures.

Five cases of extra-cerebellar tumours were also without neuritis. Two of these tumours growing on the trunk of the eighth nerve may come under the category of pontine tumours, in so far as the part of the brain chiefly affected by the growth of these tumours was the pons. Of the remaining three, two were extra-dural tumours and did not involve the cortex of the cerebellum at all. The fifth case was a large tumour in the left posterior fossa pressing on the pons and medulla and cerebellum. The left disc in this case showed some pallor, though no evidence of neuritis.

Excluding multiple tumours, there are 100 cases of tumours involving the cerebral hemispheres. Of these, sixty-eight were cortical and thirty-two deep or subcortical tumours. Of the sixty-eight tumours affecting the cerebral cortex, all those in the tempero-sphenoidal lobe had optic neuritis. With one case of a frontal tumour in which pressure atrophy developed I have already dealt. There remain seven cases in which no neuritis manifested itself, and those seven are divided between the precentral and postcentral regions. In Case 13 the tumour, though at the time of death it involved the whole of the left frontal lobe except the orbital plate, and even pressed across into the left frontal lobe, had obviously originated deep in the hemisphere and had grown down into the basal nuclei as well as up into the frontal cortex. This patient presented none of the classical symptoms of tumour, having neither headache, vomiting, nor neuritis. The other three cases of precentral tumour without neuritis were of meningeal origin, and in two certainly, and in the third probably, the brain substance was not invaded. There are no characteristics which enable us to differentiate the three cases of post-central tumour without neuritis from those with neuritis. But, as will be seen later on, the neuritis associated with tumours in this region is often very late in developing, very slight in severity, and short in duration. Further, valuable localizing signs are obtainable frequently early in the history of such cases, so that operative measures may be taken without awaiting the development of changes in the optic disc.

Subcortical tumours show the largest percentage of cases without neuritis, fourteen out of thirty-two. Of the remaining eighteen, five showed only very slight neuritis:—*Case* 69.—The neuritis lasted only a few weeks and subsided without leaving any traces. *Case* 81: Slight blurring of right disc with a little hyperæmia. *Case* 82: No neuritis was present two days before death, disc-changes were found *post mortem.* *Case* 87: Never amounted to a measurable swelling. And *Case* 91: Swelling not more than one dioptre at any time in either eye. In any one of these five cases the existence of neuritis might quite easily have passed unobserved. And, indeed, in three of them it is doubtful whether one is justified in applying the term at all. If we add these cases to the fourteen in which no neuritis occurred, we find that in nineteen cases out of thirty-two (60 per cent.) optic neuritis was a non-existent or insignificant symptom in the clinical history. Here it is rather the cases which get neuritis than those which escape that demand critical examination. Firstly, all the tumours which were confined to the white matter of the hemispheres escaped neuritis, though involvement of the cortex did not necessarily lead to development of neuritis. On the other hand, when growth inwards took place with involvement of the basal nuclei and of the lateral ventricles, optic neuritis almost invariably resulted, though in Case 94 all the basal ganglia were involved in the tumour without neuritis developing.

The single case of multiple tumours, Case 199, which did not develop neuritis adds strength to my argument, for the tumours were two in number—one right subcortical and the other right pontine.

There remain to be considered three cases of tumours affecting the mid-brain and optic thalamus, where optic neuritis was absent. In Cases 103 and 114 we can, I think, find an adequate explanation. In both cases there was great distension of the ventricles. The floor of the third ventricle was involved in this distention, and there was great and continuous pressure directly on the chiasma. In other words, there was here a condition of direct pressure exactly similar to that dealt with in the first part of this chapter, resulting in the same way in blindness, but without the development of any preceding œdema in the nerve-head. The third case (115) was very similar to Case 94, already mentioned, since here again the basal nuclei on the left side were all attacked by a melanotic sarcoma without neuritis appearing.

We can sum up the preceding section on the incidence of the neuritis in three statements:—

(1) When a tumour directly or indirectly exercises constant pressure on the chiasma or on the optic nerves it is likely to cause a primary pressure atrophy without any preceding œdema of the disc.

(2) Some cases of meningeal tumours where the brain substance is not invaded do not develop optic neuritis.

(3) There are two regions of the brain—the pons Varolii and the white matter of the cerebral hemispheres—in which tumours frequently develop without causing optic neuritis.

CHAPTER III.

The Severity of the Neuritis.

There are several factors which we have to consider in determining the degree of severity of optic neuritis.[1] Unfortunately, only one of those, the amount of swelling, can be expressed as a direct measurement, but under favourable conditions this particular point can be ascertained with very great accuracy. And in my experience, with rare exceptions, the height of the swelling fairly expresses the severity of the process which we call optic neuritis.

The second important feature in estimating the severity of the neuritis is the degree of venous turgescence and capillary congestion present. This forms one of the most striking elements in making up the general ophthalmoscopic picture. Dependent on this is the hæmorrhagic character of the neuritis. Along with the hæmorrhages and also dependent on the degree of venous congestion present there sometimes appear dusky, yellowish patches of exudate. An equally important component of the picture is the extent to which the neighbouring retina is affected by the spreading of the œdema from the neighbourhood of the disc. In a definite proportion of the more severe cases the presence of the spreading œdema is manifested by the formation of what is known as a macular fan. This feature is a very definite evidence of neuritis of a gross character, and never appears with the lower degrees of swelling, whereas hæmorrhages occasionally show themselves in cases of neuritis which cannot in any way be described as severe and in association with swelling of quite a small amount. The average swelling calculated from all the cases with hæmorrhages varies but little from the general average of swelling met with in all cases, whereas the general average in cases with macular figures is 0·6 dioptre higher.

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[1] I have purposely made this ophthalmoscopic description very brief. For a full and excellent description *see* Gunn [1].

I do not consider that the visual loss is to be regarded as a factor in determining the severity of the neuritic process. It is well known that many cases pass through a most severe attack of neuritis, even to the development of macular figures, without the vision being affected in any sensible degree. In a later part of the paper I consider more fully the nature of the relationship between the visual loss and the optic neuritis.

TABLE II.

Severity of Neuritis.

	Average swelling	Highest swelling observed	Cases with consecutive atrophy when first seen	Cases with hæmorrhages	Percentages with hæmorrhages	Average swelling in cases with hæmorrhages	Cases with macular figures
Precentral	4·8 D	6·5 D	2	15 out of 23	65	4·7 D	4
Postcentral	3·6 D	6·0 D	—	9 out of 22	41	4·2 D	4
Temporo-sphenoidal ...	4·6 D	6·0 D	—	8 out of 13	61	4·6 D	2
Subcortical	3·5 D	7·0 D	2	7 out of 16	44	3·8 D	—
Optic Thal. and Mid-brain ..	5·0 D	8·0 D	1	7 out of 11	64	5·3 D	2
Multiple	5·3 D	7·0 D	1	2 out of 5	—	—	—
Cerebellar	4·6 D	7·0 D	8	10 out of 25	40	4·8 D	7
Extra-cerebellar	5·0 D	6·0 D	—	9 out of 14	64	4·9 D	2
Pons	5·3 D	7·0 D	—	4 out of 9	44	5·3 D	1
Third Ventricle	—	5·5 D	—	3 out of 8	37	4·5 D	—
Fourth Ventricle	4·4 D	5·0 D	—				
Totals	4·5 D[1]	—	—	74 out of 146[2]	—	—	22 out of 146[3]

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[1] Mean swelling in 212 discs at highest observed swelling.

[2] Cases with consecutive atrophy excluded.

[3] Mean swelling of discs in 22 cases equalled 5·1 D.

There are certain points which come out in a striking fashion in an examination of this table. In the first place it is quite obvious that the neuritis is much less severe in subcortical and postcentral tumours than with those anywhere else. Not only is this so, but also in a considerable number of cases in both those groups the process was also of very short duration. A proof of its comparatively late development in the history of such tumours is afforded by the fact that out of thirty cases in which neuritis developed after admission of the patient into the hospital, ten were cases of postcentral tumour and seven were subcortical (see Table III.). On the other hand, pontine tumours, when they do develop optic neuritis, manifest it in a very severe form, and the same may be said of extra-cerebellar tumours.

Judged by the swelling alone, the severity of the neuritis in cases of cerebellar tumour would seem to be little above the average. And in this class the proportion of cases that showed hæmorrhages was not very high, yet we find a larger proportion of cases with macular figures than in any other class. The explanation of the position which this region occupies in the scale of severity seems to lie in the rapidity with which atrophy supervenes in many cases of cerebellar tumour; eight of the fourteen cases in which there was so-called consecutive atrophy at the time the patients were admitted to the hospital were cerebellar tumours. It is probable that these cases had passed through a neuritis of considerable severity during the earlier stages of tumour development, the details of which, if they had been available, would have sensibly altered the relative position of cerebellar tumours.

We arrive, then, at the following tentative conclusions based on the available material as to the incidence and severity of optic neuritis in tumours of the different brain regions:—

(1) Precentral tumours are nearly always associated with neuritis fairly severe in character.

(2) Postcentral tumours are nearly always associated with neuritis, as a rule moderate, and often of very short duration.

(3) Temporo-sphenoidal tumours are always associated with neuritis of about the same degree of severity as in frontal tumours.

(4) Of subcortical tumours about one half develop neuritis—as a rule, moderate in degree—and, as in the case of parietal tumours, frequently of short duration.

(5) Optic thalamus and mid-brain tumours are almost invariably associated with optic neuritis of very great severity.

(6) Cerebellar tumours are constantly accompanied by neuritis of a grave character.

(7) Extra-cerebellar tumours, as a rule, develop neuritis of a grave character.

(8) Of pontine tumours, about one half only develop optic neuritis, and then only when neighbouring parts of the brain, especially the cerebellum, have become involved. The neuritis when it does develop is usually very severe.

(9) Ventricular tumours develop a moderate neuritis.[1]

CHAPTER IV.

The Relationship of the Amount of Neuritis to the Side of the Tumour.

The important discussion which followed the reading of a paper by the author before the Ophthalmological Society of the United Kingdom in the early part of 1908 mainly bore on the question of the value of a difference in the amount of optic neuritis in the two eyes as indicating the side of the tumour. The writer was fully conscious that the views which he then expressed were not in accordance with the teaching of many authorities. Any statement which seeks to controvert the opinion held by men of such great experience in neurology as Sir Victor Horsley [2] and Sir William Gowers must needs have strong evidence behind it if it is to secure acceptance. The practical importance of the question can hardly be over estimated, and it is essential that the cases on which any conclusions may be based should be submitted to a rigid scrutiny.

It may be stated as a general rule that when neuritis starts in one eye earlier than in the other, only occasionally does the neuritis remain greater in the eye which first shows it; in most cases at the height of the swelling the neuritis becomes equal in both eyes. It does happen rarely that one eye may show greater neuritis in the earlier stages, and with an increasing neuritis in both eyes the opposite eye may show the greater neuritis in later stages.

In thirty cases of the present series the neuritis developed while the patients were under observation. In thirteen of those it appeared simultaneously in the two eyes. In eleven it developed first in the opposite eye, and in six only did it develop first in the eye on the same side as the tumours (*see* Table III.).

In dealing with the question of the amount of neuritis, we can only take those cases in which no signs of subsidence or atrophy had made their appearance. This will lead to the exclusion of fifty-eight out of the 160 cases in which neuritis developed, leaving 102 cases. I have put down as equal in swelling all cases in which the difference between the measurement in the two eyes does not exceed 0·5 dioptre. As will be seen from the table, the numbers come out that in fifty-four cases the swelling was equal in two eyes. In twenty-five the swelling was greatest in the eye on the side of the tumour, and in twenty-three it was greatest in the opposite eye.

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[1] In tumours of the floor of the third ventricle a simple pressure atrophy may replace the neuritis.

Intrinsic cerebellar tumours formed the class in which the eye of the same side most frequently showed the greatest neuritis. If, however, we sum up the subtentorial tumours, we find that in nine the greatest swelling was in the eye of same side, and in six it was in the opposite eye; while in the case of supratentorial tumours the numbers are sixteen for the eye of same side and sixteen for the opposite eye.

In the single case of multiple tumours, one was subtentorial and one supratentorial, but both were on the left side.

TABLE III.

Site.	Neuritis, commencing in eye of same side	Neuritis, commencing in eye of opposite side	Neuritis, commencing simultaneously in both eyes	Total	Greatest swelling in eye of same side	Greatest swelling in eye of opposite side	Swelling equal in both eyes	Tota
Precentral	1	2	2	5	4	4	11	19
Postcentral	3	3	4	10	6	3	8	17
Temporo-sphenoidal	1	—	—	1	1	1	7	9
Subcortical	—	3	4	7	2	4	7	13
Optic Thal. and Mid-brain	—	—	1	1	3	3	3	9
Multiple	—	1	—	1	—	1	3	4
Cerebellar	1	—	—	1	7	1	9	17
Extra-cerebellar	—	1	1	2	2	3	4	9
Pontine	—	1	1	2	—	2	2	4
Third and Lateral Ventricle	—	—	—	—	—	1	—	1
Totals	6	11	13	30	25	23	54	102

It is impossible to go into the details of the whole of this series of forty-eight cases, in which there was definite inequality in the amount of neuritis. But we may subdivide them into four classes according to the amount of inequality, and then deal in tabular form with the more important of these classes. The first subdivision includes three cases in which the neuritis had affected only one eye at the time of death or operation.

TABLE IV.

Neuritis affecting only one eye at the time of death or operation.

Case	Site of Tumour	Amount of Neuritis		Remarks
		Right Eye	Left Eye	
1	Right precentral	None	+ 2·0 D	Neuritis intense in character with hæmorrhages in left eye
34	Right rolandic	Haziness, no swelling	+ 1·5 D	Edges of left disc blurred
200	Left ascending frontal and left extra-cerebellar	Commencing neuritis	Normal	Neuritis early but definite in right eye

It will be seen that in each of these three cases the neuritis at the time of death or of operative interference had appeared definitely in one eye only, and in each case it was the eye on the side opposite to the tumour which showed the neuritis. To the first case I have already referred in the early part of Chapter II., where I have given what I think is the true explanation of the non-development of the neuritis in the right eye. In the other two cases the neuritis, though of short duration, was perfectly definite in character.

The second subdivision consists of cases which showed a difference of two dioptres or more between the highest measured swelling in the two eyes.

TABLE V.

Cases showing 2 D *or more difference between the swelling in the two eyes.*

Case	Site of Tumour	Amount of Neuritis		Remarks
		Right Eye	Left Eye	
3	Right Precentral ...	1 D	4 D	Vision very bad before neuritis
22	Right Precentral ...	4 D	6 5 D	Neuritis equal in earlier stages
47	Left Postcentral ...	3 D	commencing	Slight blurring in left eye three days before death
73	Left Subcortical ...	3 D	1 D	Neuritis developed first in right eye
113	Mid-brain, mostly left side	8-10 D	7 D	Very intense neuritis. Tumour much greater on left side than on right
154	Left Cerebellar ...	6·D	3 D	—
185	Left lateral Ventricle and third Ventricle	5·5 D	3·5 D	
40	Right Postcentral ...	2 D	commencing	Swelling measurable in right eye only on day of death
42	Right Hemisphere ..	3·5 D	1 5 D	Large extra-cerebral blood-cyst
101	Right Optic Thalamus	6 D	4 D	
153	Left Extra-cerebellar	3 D	5 D	

The first seven cases in this table show a very much greater amount of swelling in the eye away from the side of the tumour than in the eye on the same side. In the last four cases it will be seen that it was the eye on the side of the tumour which had the greater swelling. In all these cases, apart altogether from the question of measurement, there were definite differences in the ophthalmoscopic pictures presented by the two eyes.

The third subdivision includes five cases in which the difference in the swelling between the two eyes was 1·5 dioptre. In four of these it was the eye on the side of the tumour which had the greatest swelling, and in one it was the opposite eye. Taking these first three subdivisions together, there are eight cases in which the eye on the side of the tumour showed the greatest swelling and eleven cases in which the opposite eye showed the most.

In the remaining cases the difference amounted to one dioptre only, and in this subdivision there were seventeen cases where the eye on the side of the tumour showed most swelling as against twelve where the opposite eye was most affected, making up the totals, as seen on Table III., to twenty-five and twenty-three respectively.

In a small number of cases the neuritis took on a hæmorrhagic character in one eye only, and while I do not lay any great stress on hæmorrhages as an indication of severity of neuritis, yet I give the numbers as showing that even in this characteristic it was occasionally the opposite eye which showed hæmorrhages. In twelve cases these appeared in the eye of the side of the tumour, and in five cases in the opposite eye.

As I have already said, there is reason to doubt the close relationship between the visual result and the neuritis, but we do find a few cases in which the vision was much more affected in one eye than in the other as the neuritis subsided. There were nine such cases, and in four it was the eye on the side of the tumour in which the vision was most affected, and in five it was the opposite eye.

In concluding this section, I may for a moment intrude a personal statement. Before I started to analyse these statistics, I should have been prepared to state, as my general impression, that the neuritis was greater on the side of the tumour. The few exceptions that I had seen—*e.g.*, Case 1—were, as I thought, capable of simple explanation. But, as the exceptions increased in number, I was forced to realize that the point was not one that could be regarded as of value in localization. Even if the number of cases where the opposite eye is the more affected were much smaller than it is, so long as we are unable to explain why in some cases it should be more affected we cannot assume that a difference in the amount of neuritis is a sign of localizing value.

CHAPTER V.

Nature of Tumour in Relationship to Neuritis.

The nature of the tumour was ascertained in 150 cases. Of these, twenty-nine had no neuritis (19·3 per cent.), so that, on the whole, they may be taken as making a fairly representative collection. An examination of the cases in detail does not lend much support to the idea that the nature of the tumour plays any important part in determining the occurrence of optic neuritis except in so far as the nature of the tumour may determine the locality. Gliomata formed a very considerable proportion of the whole, there being sixty-three cases. Of these, thirteen had no neuritis, so that in this case at any rate the percentage, 20 to 63, without neuritis is the same as we find in tumour cases in general.

TABLE VI.

Nature of Tumour	Glioma		Sarcoma		Cysts		Endothelioma		Tubercle		Fibroma		Carcinoma		Gumma		Papilloma		Total	
	Neuritis present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent	Present	Absent
Frontal	13	1	—	2	1	1	5	1	—	—	1	—	1	—	1	—	—	—	22	5
Parietal	4	—	2	—	4	—	1	—	—	—	—	—	—	1	1	—	—	—	12	1
Temp.-sphen.	3	—	4	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	9	—
Subcortical ...	3	7	2	—	3	—	1	—	—	—	—	—	—	—	—	—	—	—	9	7
Optic Thal. and Mid-brain ..	6	—	1	2	—	—	—	—	2	1	—	—	—	—	—	—	—	—	9	3
Multiple ...	—	—	—	—	—	—	2	—	—	—	—	—	4	—	—	1	—	—	6	1
Cerebellar .	11	—	5	—	6	—	—	—	4	—	—	—	—	—	1	—	—	—	27	—
Extra-cerebellar	1	1	1	2	—	—	1	1	—	—	9	—	—	1	—	—	—	—	12	5
Pontine ..	5	4	1	1	—	1	—	—	2	—	—	—	—	—	—	—	—	—	8	6
Vermicular ...	4	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	2	—	7	1
Totals	50	13	16	7	14	3	12	2	9	1	10	—	5	2	3	1	2	—	121	29
Percentage without neuritis...	20·63		26.09		17·64		14·28		10		—		28·57		25		—		19·3	

In some of the other cases the numbers are too small to afford a trustworthy basis for percentage classification, but I give the list in the inverse order of the frequency of neuritis.

No Neuritis.

Carcinoma	2 out of 7 cases	—
Sarcoma	7 out of 23 cases	26·1 per cent.
Gumma	1 out of 4 cases	—
Glioma	13 out of 63 cases	20·6 per cent.
Cysts	3 out of 17 cases	17·6 per cent.
Endothelioma	2 out of 14 cases	14·3 per cent.
Tubercle	1 out of 10 cases	—
Fibroma	0 out of 10 cases	—
Papilloma	0 out of 2 cases	—
	29 out of 150 cases	19·3 per cent.

At first sight it certainly seems a little curious that this list should be headed by the two classes of tumours which show the most malignancy. But one of the two cases of carcinoma was an extra-dural tumour in the cerebellar region which did not invade the brain substance at all, and the other was quite a small tumour in the parietal region.

The elucidation of the cases of sarcomata presents greater difficulties. We find that one of the seven cases is the mid-brain tumour (Case 103) for which we have already attempted to provide an explanation of the non-occurrence of optic neuritis in the dilatation of the third ventricle; if we except this case the percentage is brought practically down to the normal. The intracranial tension in at least four of the remaining cases was high, and in one of them very high (Case 158). These sarcomatous tumours were not limited to any one region. When we come to the other end of the list we find that practically all the cases of tubercle were associated with optic neuritis. The explanation of this seems to be that the tuberculous growths were much more frequent in the mid-brain and cerebellum than in any other part of the brain. A similar argument applies in the case of a fibromata, where nine of the ten cases described were extra-cerebellar tumours. The two cases of papilloma described were both intra-ventricular tumours and both associated with neuritis.

A critical examination of the relation existing between the nature of the tumour and the optic neuritis forces us to conclude that there is nothing in the nature of the tumour itself which determines the onset of neuritis. Not even does rapidity of growth seem to play any great part, nor size, as gross neuritis may be caused by a very small tumour in one place, while a very large tumour may be present in another place without any change appearing in either disc. In so far as the nature of the tumour determines its location, just so far and no further does it seem to determine its relationship to the neuritis.

If we examine the effect of this conclusion on the question as to the pathogenesis of the optic disc phenomena, we see that, in so far as it goes, it is against the toxin hypotheses, since the rate of breaking down of brain matter and the rapidity of development of toxins will not in any way depend on the position of the tumour, but will depend on its nature and on the rapidity of its growth. For example, gliomata tend to break down readily, and yet, as we have seen, they do not proportionately cause a greater amount of optic neuritis, while fibromata show little tendency to break down themselves or to cause breaking down of brain substance, and yet most of them are associated with neuritis.

CHAPTER VI.

The Relationship of Optic Neuritis to Intracranial Tension and to the occurrence of Meningitis.

Evidence of the state of intracranial tension during life is only obtainable directly in cases which are submitted to operation. There is little doubt that the tension varies considerably from time to time, and that the attacks of increased headache, giddiness, vomiting and temporary dimness of sight coincide with periods of raised tension. The reports of the amount of intracranial tension at the time of operation depend on the personal equation of the observer, and any distinction between cases must be, to a considerable extent, an arbitrary one.

Post-mortem evidences must depend to almost as great an extent on the duration of the raised tension as on the amount of it. *Post-mortem* evidences will also vary with the position of the tumour. Thus a supratentorial tumour will be more likely to show much flattening of the cerebral convolutions when the tension has been raised for some time, while a subtentorial tumour will be more likely to show a large pressure cone in the foramen magnum.

In a considerable number of cases there is unfortunately no indication in the notes as to the condition of the intracranial tension. I have attempted to subdivide the other cases into three classes.

Class I. showed evidences of very high intracranial tension—*e.g.*, great bulging of the dura at the time of operation or a very marked pressure cone or much flattening of the convolutions or much distended ventricles at the autopsy.

Class II., cases which gave definite evidence of raised tension at operation or *post mortem*, though not of such a gross character as in Class I.

Class III., where the operation notes definitely state that the tension was not raised or where the *post mortem* revealed no flattening of the convolutions nor distension of the ventricles.

TABLE VII.

Intra-cranial Tension Cases with Neuritis	Very high	High	Slight or normal	
Precentral	3	6	1	10
Postcentral	2	6	1	9
Temporo-sphenoidal	3	7	1	11
Subcortical	1	12	—	13
Multiple	—	—	1	1
Optic Thalamus and Mid-brain	5	6	1	12
Cerebellar	11	14	3	28
Extra-cerebellar	4	5	1	10
Pontine	5	1	—	6
Third Ventricle	2	1	—	3
Fourth Ventricle	2	3	—	5
	38	61	9	108

TABLE VIII.

Intra-cranial Tension Cases without Neuritis	Very high	High	Slight or normal	
Precentral	—	3	—	3
Postcentral	—	1	—	1
Temporo-sphenoidal	—	—	—	—
Subcortical	1	2	2	5
Multiple	—	1	—	1
Optic Thalamus and Mid-brain	2	1	—	3
Cerebellar	—	—	—	—
Extra-cerebellar	1	1	—	2
Pontine	—	1	5	6
Third Ventricle	—	—	1	1
Fourth Ventricle	—	—	—	—
Totals	4	10	8	22
Percentage of Total Cases in each class.	9·523 %	14·084 %	47·05 %	

Taking the tables as a whole, they seem to favour the hypothesis that the occurrence of neuritis is closely related to the amount of intracranial tension, but I do not think we have at the present time evidence enough to justify us in saying that the relationship is one of cause and effect. It is possible that the raised intracranial tension and the optic neuritis are two concurrent results of one common cause. For instance, a change in intracranial conditions due to the presence of the tumour may cause an œdema of brain tissue which, in course of time, spreads to the optic nerve, and at the same time may play a part in raising the intracranial tension.

Table VIII. shows four cases in which there was very high tension without optic neuritis. I have already dealt in detail with the two mid-brain cases (103 and 114) in Chapter II., and have offered an hypothesis to explain the non-appearance of neuritis, despite the very high tension. The other two cases (86 and 158) remain unexplained exceptions to the rule, that very high intra-cranial tension is accompanied by optic neuritis.

Pontine tumours are of special interest in this connection. I have laid great stress on the conditions under which these tumours produce neuritis, and a comparison of Tables VII. and VIII. shows that the appearance of neuritis and a great rise in intracranial tension are always associated. In each table there are six cases. Five of those with neuritis had very high tension, five without neuritis had normal intracranial tension. It is much to be regretted that we cannot in a similar manner contrast the cases of subcortical tumour with and without neuritis, but, unfortunately, in nine out of the fourteen cases without neuritis the notes do not afford any evidence as to the condition of the intracranial tension. One would like to suppose that this was because the tension was not raised. To make such a supposition in this case would involve doing the same in the larger number of cases of cortical tumour (precentral and postcentral) with neuritis in which notes of intracranial tension are equally lacking.

The possibility of optic neuritis being due to an inflammation spreading from the meninges down the nerve-sheaths may be dealt with very briefly. It must be understood that the description of the meninges in the reports is in the large majority of cases based only on the macroscopic appearances. In twenty-eight cases only was there any meningeal change noted. In twelve of these this consisted of nothing more than inflammatory adhesions confined to the surface affected by the tumour. In seven cases there was post-operative meningitis which could have had nothing to do with the development of the neuritis, and, indeed, in four cases in which the meningeal inflammation was most generalized there was no neuritis. In the great majority of *post-mortem* reports the meninges were stated to be healthy.

CHAPTER VII.

The Vision in Cases of Cerebral Tumour.

I have stated in an earlier part of this paper that there is grave reason to doubt that the loss of vision that occurs in cases of cerebral tumour is due to the condition which we call optic neuritis. Further, we may ask the question whether the atrophy which appears later in the history of the case is really consecutive on this process or is a simple pressure atrophy. Before I proceed to the consideration of this, there are one or two points about the vision with which I wish to deal. In some cases, of course, the mental condition of patients precluded the possibility of accurate subjective testing at any time during the history of the case. In other cases the vision could be tested before operation, but the effect of the operation itself on either the vision or the neuritis could not be noted.

There were 101 cases with neuritis in which a fairly full history of the state of the vision could be obtained, and of the cases without neuritis the vision could be tested in a large proportion.

TABLE IX.

Visual Results—Operation Cases	$\frac{6}{6}$—$\frac{6}{12}$	$\frac{6}{18}$—$\frac{6}{36}$	$\frac{6}{60}$— finger counting	P.L.—blind	One eye bad vision		Vision defective for reasons apart from Neuritis
					Same side	Opp. side	
Precental	7	1	—	1	1	1	1
Postcentral	6	4	2	2	1	—	2
Temporo-sphenoidal	—	1	—	—	1	—	—
Subcortical	4	—	—	—	—	—	—
Optic Thalamus and Mid-brain	—	—	—	3	—	—	—
Cerebellar	7	1	—	4	1	2	—
Extra-cerebellar	3	—	1	—	—	—	—
Pontine	—	—	—	—	—	—	—
Ventricular	—	—	—	—	—	—	—
Totals	27	7	3	10	4	3	3
	[1]57·44%	14·9 %	8 %	21·27 %			

$$\underbrace{\underbrace{57{\cdot}44\%\quad 14{\cdot}9\%}_{{}^{2}34\,=\,72{\cdot}34\,\%}\qquad \underbrace{8\%\quad 21{\cdot}27\%}_{13\,=\,27{\cdot}66\,\%}}_{47\text{ Cases.}}$$

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[1] Fair vision, 22 out of 30 = 73·3 %
[2] Good vision, 18 out of 30 = 60 %

TABLE X.

Visual Results—Non-Operative Cases	$\frac{6}{6}$—$\frac{6}{12}$	$\frac{6}{18}$—$\frac{6}{36}$	$\frac{6}{60}$—finger counting	P.L.—blind	One eye bad vision		Vision defective for reasons apart from Neuritis
					Same side	Opp. side	
Precentral	—	1	1	1	—	—	1
Postcentral	2	1	—	—	—	—	—
Temporo-sphenoidal	2	2	—	1	—	—	—
Subcortical	4	1	1	1	—	1	—
Optic Thalamus and Mid-brain	—	1	1	2	—	1	—
Cerebellar	1	—	2	4	—	—	—
Extra-cerebellar	—	1	1	1	—	—	—
Pontine	1	1	—	3	—	—	—
Ventricular	2	—	1	1	—	—	—
Totals	12	8	7	14	—	2	1
	29·27 %	19·51 %	17·07 %	34·14 %			

$$\underbrace{\underbrace{29{\cdot}27\ \%\quad 19{\cdot}51\ \%}_{20\ =\ 48{\cdot}78\ \%}\quad \underbrace{17{\cdot}07\ \%\quad 34{\cdot}14\ \%}_{21\ =\ 51{\cdot}22\ \%}}_{41\ \text{Cases.}}$$

The first point I wish to deal with is the beneficial effect of operation on vision. For this purpose we must exclude the cases in which atrophy was obvious before the patient came under observation. There remain eighty-eight cases, forty-seven of which were operated on and forty-one were not.

These tables show how very beneficial operation is in saving sight. Table IX. gives a larger number of cases than I gave in my paper in Volume XXV. of the *Transactions of the Ophthalmological Society*, but the percentages remain unchanged. The vision in Table X. is the last recorded before death. As I am dealing in the present paper with confirmed cases alone, and as the confirmation of a case not operated on can only take place after death, it is not right to make too many inferences from a comparison of these tables. As regards Table IX., there is one point of very considerable practical importance. Of the forty-seven cases mentioned in it, twenty-eight were alive when last heard of, many of them in perfectly good health. If we distribute these into their respective classes as regards vision, we find that of the twenty-seven with good vision ($\frac{6}{6}$ to $\frac{6}{12}$), nineteen are still alive; of the seven with moderately good vision ($\frac{6}{18}$ to $\frac{6}{36}$), six are still alive; so that of thirty-four cases operated on retaining moderately good vision, twenty-five are alive, while only three of the thirteen with bad vision are alive. This, I think, has an important bearing on the question of the time to choose for operation in such cases, since it would seem to be obvious that if operation is deferred till visual loss is well marked, the chance of the patient surviving is very much diminished. I return to this point later on.

In Table XI. I give the visual results in cases with optic neuritis as they appeared in tumours of different parts of the brain.

TABLE XI.

Visual Results in cases of Optic Neuritis, excluding cases which died within a few weeks of operation or whose vision could not be taken [but including cases of consecutive atrophy when first seen].

	Vision $\frac{6}{6}$—$\frac{6}{36}$	Vision $\frac{6}{60}$—blind	Percentage of cases vision badly affected
1. Optic Thalamus and Mid-brain	1	7	87·5
2. Cerebellar	9	18	66·6
3. Pontine	2	3	60·0
4. Ventricular	2	2	50·0
5. Extra-cerebellar	4	3	42·85
6. Frontal	9	5	35·71
7. Subcortical	9	4	30·77
8. Parietal	13	4	23·53
9. Temporo-sphenoidal	5	1	16·6
Totals	54	47	46·53

This table shows that the tumours which are most likely to cause ventricular distension are those which cause the greatest loss of vision.

I wish to suggest as a possibility—worthy, at any rate, of being seriously considered—that the affection of vision which takes place in cerebral tumours is not of necessity the result of the affection of the nerve-head which we indiscriminately term papillitis, optic neuritis, or choked disc. There are several reasons which can be adduced in favour of regarding the visual loss and the papillary œdema as more or less independent of one another. (1) There is the undoubted fact that many cases go through a most severe attack of optic neuritis without the visual acuity being affected in the slightest degree. (2) I pointed out in a previous paper that attacks of temporary amaurosis are frequently met with in cases of cerebral tumour, that these attacks are always associated with increased headache and giddiness, that they may be met with in cases in which no disc-changes were manifest, and did occur in three cases in which no neuritis ever did develop. (3) I have already spoken of two cases of tumours in the mid-brain which did not develop any optic neuritis. One of these cases (H. F.) had become quite blind within about four weeks, though the discs were normal in appearance. Of the other, the mental condition did not allow the vision to be tested, but the pupillary reflex to light was completely lost. (4) In certain cases in which neuritis did develop, the failure of vision distinctly preceded the appearance of any disc-changes. (5) The vision is most affected in cases in which there is the greatest likelihood of distention of the third ventricle [compare visual table (XI.) with tables of intracranial tension (VII. and VIII.)]. (6) The recovery of vision after operation in many cases takes place at a very much more rapid pace than the subsidence of the neuritis. (7) In certain cases of the present series we have evidence that direct pressure of the tumour on the nerve or the chiasma causes blindness, *e.g.*, and this blindness may exist for some time before signs of descending atrophy manifest themselves in the disc. (8) Frequently cases which have been seen to pass through an attack of papillitis which has completely subsided present a condition of disc which it is practically impossible to distinguish from the disc of a primary atrophy.

These points, I think, must suggest that the visual loss and the subsequent atrophy may, at least in some cases, depend on some factor other than the papillary changes, and I would suggest the possibility that that factor is the direct pressure of the distended floor of the third ventricle on the chiasma. And I think this will account for the fact that the vision is less affected in temporo-sphenoidal tumours than in any other class of case, though in all other respects temporo-sphenoidal tumours stand so high up in the degree to which they are affected by optic neuritis; and it will also, I think, account for the bad visual results in all the cases in which there is a likelihood of blockage of the aqueduct of Sylvius.

If we accept this hypothesis provisionally as it affects the sight, it may be that the atrophy which appears in the later stages of this process is also to be regarded as analogous to any other pressure atrophy, only modified by the co-existence of an œdema in its earlier stages, and if this is so we may have to look to the chiasmal end of the optic nerves rather than the disc end for the earliest signs of the atrophic process. The point is one of great practical importance, since in many cases it is necessary to perform a decompression operation to save sight where no definite localization of the tumour can be made; and, if this hypothesis be correct it is the commencement of visual failure that should be taken as an indication for palliative trephining rather than the occurrence of optic neuritis. All the evidence goes to show that if cases are operated on while vision is little affected, the chances for the patient surviving are good, and this would seem to be true even when the neuritis has been in existence for a prolonged period. On the other hand, the worse the vision the poorer is the patient's chance of surviving. This, however, may simply be due to the fact that it is in mid-brain and subtentorial tumours that the vision is most likely to be seriously affected.

CHAPTER VIII.

The Age-incidence of Optic Neuritis in Cerebral Tumours.

Before we can properly discuss the question of whether optic neuritis diminishes in frequency and in severity with increasing years, we must first see what the age-incidence of tumours in general is. The cases in the present series varied in age from 1½ years up to 68, but the enormous majority of the cases were between 15 and 50. The average age of all the cases was 34. Table XII. gives the age-incidence of the tumours in general, apart from any question of neuritis.

TABLE XII.

Age-incidence of Tumours in different regions of the Brain.

Site of Tumour.	Average age.	Cases under 30.	Cases between 30 and 40.	Cases over 40.
Precentral	37	9	12	8
Postcentral	38	5	9	10
Temporo-sphenoidal	34	4	4	4
Subcortical	41	7	9	16
Optic Thalamus and Mid-brain ...	27	9	3	2
Cerebellar...	15	27	3	2
Extra-cerebellar	39	5	6	8
Pontine	15	12	—	3
Ventricular	36	4	1	4
Multiple	46	1	2	4
Totals	—	83	49	61

If we take the mean of the ages of patients with neuritis we find that it is 32, two years less than the mean age for all tumours. On the other hand, if we take the mean age of cases without neuritis, it is 40, six years higher than the general average. An examination of Table XIII. will, I think, serve to show that this does not really mean that optic neuritis tends to become less frequent because of advancing age. The true explanation lies in the fact that all cerebellar tumours get optic

TABLE XIII.

Age-incidence in Tumours with and without Neuritis.

Site.	With Neuritis.			Without Neuritis.		
	Cases under 30	Cases between 30 and 40	Cases over 40	Cases under 30	Cases between 30 and 40	Cases over 40
Cortical	17	20	18	—	3	3
Subcortical	4	6	8	3	4	9
Optic Thalamus and Mid-brain ...	7	3	1	2	—	1
Cerebellar	27	3	2	—	—	—
Extra-cerebellar	2	6	6	2	1	2
Pontine	9	—	—	3	—	3
Ventricular	4	1	3	—	—	—
Multiple	1	1	4	—	1	—
Totals	71	40	42	10	9	18

neuritis, and cerebellar tumours are more particularly prone to occur in the early years of life, as shown in Table XII., whereas subcortical tumours, which frequently escape without neuritis, are much more frequent in later life. A comparison of the two parts of Table XIII. reveals how much the different age-incidence in the two classes depends on the one hand, on the preponderance of cerebellar tumours, and on the other on the preponderance of subcortical tumours. And I think it must be admitted that, apart from these two regions, age-incidence of cases with neuritis and without neuritis would be very similar. The conclusion which I have come to from a careful examination of this series of cases is that age in itself has little or no influence in determining the onset of neuritis.

The age-incidence of cases which showed hæmorrhages corresponded almost exactly with that of the cases with neuritis, the average age being 33. The average age of the cases with macular figures is definitely lower—27 years. The fact that the cerebellar cases form such a definite proportion with this change would have led us to anticipate some such result.

CHAPTER IX.

Summary of Conclusions.

I may briefly recapitulate the main conclusions which the examination of this series of 200 cases has led me to:—

(1) Tumours directly or indirectly exercising constant pressure on

the chiasma, or on the optic nerves, may cause primary pressure atrophy without any preceding œdema of the disc.

(2) The great majority of tumours affecting the grey matter of the cortex cerebri have optic neuritis, the severity of which seems to vary inversely with the distance of the part affected from the chiasma.

(3) Tumours lying deep in the white matter of the cerebral hemispheres do not, as a rule, develop optic neuritis until they invade either the cortex or the basal ganglia. They are most likely to develop optic neuritis when they spread in the latter direction.

(4) Tumours affecting the optic thalamus, the mid-brain, the cerebellum and the ventricles are almost invariably associated with optic neuritis.

(5) Pontine tumours and some extra-cerebellar tumours which are closely allied to pontine tumours do not develop optic neuritis until neighbouring regions, especially the cerebellum, are invaded.

(6) A difference in the intensity of the neuritis in the two eyes is met with in about 50 per cent. of all cases. The neuritis in some cases is greater in the eye on the side of the tumour, and in some cases is greater in the opposite eye. When a difference exists it should not be regarded as of localizing value.

(7) The nature of a tumour does not in itself play any part in determining the onset of neuritis, except in so far as the nature of the tumour determines its location.

(8) A close relationship seems to exist between the occurrence of neuritis and the amount of intracranial tension, but there is as yet nothing to justify us in saying that the relationship is one of cause and effect.

(9) Meningeal inflammation is rare in cerebral tumours, and when it does occur is, in the majority of cases, localized to the actual tumour area and plays no part in the causation of optic neuritis.

(10) There are reasons for thinking that the diminution of visual acuity and the œdema of the optic nerve-head are more or less independent of one another, and are not related to one another as cause and effect.

(11) The atrophy of the nerve in some cases at least is probably a simple pressure or descending atrophy starting from the chiasmal end of the nerve and exactly analogous to the primary atrophies that occur quite apart from any œdema of the disc.

(12) Age in itself has little or no relationship to the occurrence of neuritis.

The desire for brevity has given a greater appearance of dogmatism to these twelve conclusions than I would wish to convey, but I can only hope that readers will bear in mind that they are based on this particular series of cases alone, and I have little doubt that future work will introduce many modifications and qualifications into these conclusions.

LITERATURE.

[1] GUNN, MARCUS. *British Medical Journal*, October 26, 1907.
[2] HORSLEY, SIR VICTOR. Presidential Address, *British Medical Association*, 1906, and Oxford Address, July, 1908. *Ophthalmoscope*, September, 1908, p. 658.
[3] PATON, LESLIE. *Trans. Ophthalmological Soc.*, 1905, vol. xxv., p. 129.

PUBLICATIONS RECENTLY RECEIVED.

[*Notes on a book under this heading do not preclude a subsequent review.*]

Ueber die Kerne des Menschlichen Rückenmarks. Von L. JACOBSOHN. S. 72, mit 9 Tafeln. Berlin: Königl. Akadamie der Wissenschaften, 1908.

This monograph gives a detailed description of the arrangement of the nerve cells in the grey matter of the spinal cord, which the author has studied in serial sections. The typical arrangement in each segment is illustrated by drawings and described in the text in much the same manner as the same subject was treated by Bruce in his well-known "Atlas of the Spinal Cord." In Jacobsohn's work, however, more attention is given to the cells of the dorsal horns and to the irregular groups which lie elsewhere in the grey matter. Unhappily these cells have received as yet very little attention in the investigation of spinal cords affected by different pathological conditions, and if this monograph succeeds in exciting interest in them it will undoubtedly repay the enormous amount of labour expended on it.

Einführung in die Histologie und Histopathologie des Nervensystems. Von PAUL SCHRÖDER. S. 101. Jena: Gustav Fischer, 1908.

An excellent introduction to the study of the structure and pathology of the nervous system. The first four chapters, forming a half of the whole book, are devoted entirely to its normal histology, the rest to the principles of neuropathology. The manner in which most of the subjects are treated is admirable; the descriptive reviews, for instance, of the structure of the neuroglia and of the lymph supply of the central nervous system are remarkably clear and concise. As the author admits in his preface, his views have been largely determined by Nissl's teaching, a fact that should make this small book very valuable to English readers, from whom, on the whole, Nissl's work has scarcely received the attention it deserves.

Beiträge zur Pathologie des Stoffwechsels bei Psychosen (Erster Teil: "Die progressive Paralyse"). Von Dr. MAX KAUFFMANN. Pp. 188. Jena: Gustav Fischer, 1908.

A careful investigation of the chemical changes in several cases of general paralysis of the insane under varying conditions. Patients were chosen in various stages of the disease, and the weight, the nitrogen taken in and given out, and the water were determined. A case of general paralysis with aseptic fever, which the author believes to be caused by defective adaptation to sudden oxidation changes, and one where the patient was attacked with erysipelas were examined daily over considerable periods. A chapter is devoted to the condition of the blood and of the cerebro-spinal fluid obtained by lumbar puncture.

Beiträge zur Pathologie des Stoffwechsels bei Psychosen (Zweiter Teil: "Die Epilepsie"). Von Dr. MAX KAUFFMANN. Pp. 199. Jena: Gustav Fischer, 1908.

The same author investigated the nutrition in five cases of epilepsy by the same methods as he employed in cases of general paralysis of the insane. He also examined the blood and the cerebro-spinal fluid. A number of interesting observations are given, but he arrived at no definite conclusions with regard to the chemistry of this disease and its manifestations.

Reports from the Pathological Laboratory of the Lunacy Department of the New South Wales Government. Vol. I. Part III. With numerous figures. Pp. 167. Published by authority of the Government of the State of New South Wales. Sydney: W. A. Gullick, 1908.

This volume contains a study of the morphology of the brain of the Australian aboriginal by Dr. Flashman, and of the skull of the Australian aboriginal by Professor Hermann Klaatsch, of Breslau. Dr. Flashman's paper gives a naked-eye description of two brains, but the greater portion of the volume is taken up by the important monograph of Professor Klaatsch, illustrated with over a hundred photographs.

Mendel's Principles of Heredity. By WILLIAM BATESON, M.A., F.R.S. With 6 coloured plates and other illustrations. Pp. 396. Cambridge: University Press, 1909.

This book gives an account of discoveries in regard to heredity made by the application of Mendel's method of research. A translation of Mendel's two papers is appended, together with a biographical note. The case for Mendel's theory is stated with all Professor Bateson's well-known clearness of exposition, and its complexities are reduced to the simplest possible form. This book should be on the shelves of every medical man interested in the problems of heredity.

Einführung in die Lehre vom Bau und den Verrichtungen des Nervensystems. Von Prof. D. Ludwig Edinger. Mit 161 Abbildungen und 1 Tafel. S. 190. Leipzig: Vogel, 1909.

This is an admirable summary of our certain knowledge of the structure of the central nervous system set out clearly for students. The descriptions are clear and simple and the figures excellent. Any physician interested in neurology would find this little book an excellent means of refreshing his knowledge of the nervous system.

Diseases of the Nervous System for the General Practitioner and Student. By Alfred Gordon, M.D. (Paris), A.M. With 136 illustrations. Pp. 487. London: H. K. Lewis, 1908.

An elementary text-book in which diseases of the nervous system are treated in the conventional manner. Most of the figures are taken from other books.

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EDITOR.

[AUGUST, 1909.]

BRAIN.

PART II., 1909.

Original Articles and Clinical Cases.

ON THE STRUCTURE AND FUNCTIONAL RELATIONS OF THE OPTIC THALAMUS.

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SYNOPSIS.

- INTRODUCTION.
- HISTORICAL.
  - Physiology.
  - Anatomy.
    - Thalamic Nuclei.
    - Fibres between Thalamic Nuclei.
    - Thalami-cortical Fibres.
    - Cortici-thalamic Fibres.
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    - Nucleus Anterior.
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    - Centre Median and Nucleus Arcuatus.
    - Hypothalamus.
- STIMULATION EXPERIMENTS.
- SYMPTOMS.
- SUMMARY.

AT the very outset I wish to express my deep gratitude to Sir Victor Horsley for the privilege of working in his laboratory, for his innumerable kindnesses, and for the benefit of his many valuable suggestions, frank criticism, and unerring judgment. He suggested the subject and method of the investigation and co-operated in the performance of the experiments and revision of the preparations. I am also much indebted to Dr. R. H. Clarke for his ever-ready assistance and advice, as well as for the use of his stereotaxic instrument. To Miss E. Clarke's unflagging interest and careful assistance the results obtained are in great part due.

The optic thalamus, with its adjacent ganglia, has been the subject of numerous investigations during the past thirty years. These have dealt with the anatomical as well as the physiological side of the question. There still remain, however, many obscure points which make this a profitable field, and particularly for a new method such as has been employed for the present work.

HISTORICAL.

The very comprehensive monograph by Roussy [135] on the optic thalamus, published during the past year, deals with the historical survey of our present state of knowledge so thoroughly that, for the sake of brevity, but a general outline of methods and results of past work will be given here.

Prior to the work of Nothnagel [115] and Meynert [104] very crude experiments were performed on the basal ganglia by Luys [95], Schiff [139], Burdach [26], Magendie [96], Serres and Loustan [143], and others.

With Nothnagel began the period of studying this region by means of localized lesions, and that plan has been followed for the most part up to the present day, though more refined methods have been employed.

These methods may be grouped under three headings:—

First.—The injection of fluids (Nothnagel and Fournié) [58].

Second.—Incisions or destruction by some mechanical means. Of these, that employed originally by Veysierre [153] and later by Probst [123], viz., a trochar containing a hidden wire which could be pushed forward to produce the lesion, was the most generally used. By the mechanical method results were obtained by Ferrier and Turner [49], Bechterew [8], Mislawski [13], Ernst [47], Probst [123], Ziehen [159], Ott [119], and others.

Third.—Electrolytic lesions, first used by Sellier and Verger [140], then by Roussy [135], and Horsley and Clarke [75], in their work on the cerebellum.

Physiology.

The earliest observers—Magendie [96], Flourens [54], and Schiff [139]—were of the opinion that the thalamus was the centre for voluntary motor functions, the last named even believing it controlled intestinal movements. These early investigators described "mouvements de manège" as characteristic of thalamic lesions.

Carpenter, in 1851, and after him Meynert [104], in the Seventies, however, expressed the following view: "The optic thalamus is the centre for unconscious reflex movements resulting from peripheral stimuli, and from it these are carried to the cortex, producing images of these movements, which then are the basis for voluntary movements." Nothnagel [116] and Ferrier [48] believed it was a sensory station between the lower centres and the cortex. Carville and Duret [31] held similar views. There followed then numerous investigations by Bechterew and his pupils—Mislawski [15-17], Ostankow, Irwanow [77], and Ernst [47]. In these experiments the cortex was removed in part and the various regions stimulated with the faradic current. They described numerous centres in the thalamus controlling various functions, movements of the stomach and intestine, bladder, vagina and rectum, also lachrymation and a screaming centre, described by Irwanow, referred to by Bechterew as a centre where stimulation produced "Geschrei wie es auf Schmerz auftritt." These investigations have, as far as can be discovered, never been confirmed.

Thiele [149] obtained locomotor movements of the limbs on stimulating the cut surface of the posterior part of the thalamus.

Hale White [68] and Ott [119] found changes in temperature associated with thalamic lesions, and the latter also noted changes in blood-pressure. Christiani described a mastication centre.

Sellier and Verger [140] found hemianæsthesia in their animals after thalamic lesions, but advanced the important conclusion that visual symptoms were not obtained from the anterior and lateral parts of the thalamus, but only from the posterior portions. Up to this time ocular symptoms, especially hemianopsia, had been described as a symptom of thalamic lesions irrespective of the region injured.

Von Monakow [107], in his elaborate monograph—the important anatomical features in which will be taken up later—regards the thalamus as wholly a sensory centre.

Probst [129], in one experiment, found slight sensory changes—hemianopsia and a paresis of the limbs—on the same side; as well as a loss of the sense of position of the extremities.

Prus [130] dividing the thalamus arbitrarily into four parts—anterior, middle, central, and lateral—stimulated each *after removing the cortex*, and concluded that the thalamus contained centres for respiration, pulse-rate, blood-pressure, motor movements, and particularly mimicry and anger. It seems an obvious criticism of these experiments that the large area of cut nerve fibres exposed after the cortex is removed may be a cause for some, at least, of the phenomena observed quite irrespective of the stimulation.

Roussy [135], after thalamic lesions, observed :—

(1) Mouvements de manège; (2) superficial sensory changes, hypoæsthesia, hypoalgesia, hypothermæsthesia; (3) deep sensory loss, evident by the loss of the sense of position of the limbs, and in the case of a monkey by a tendency to use the unaffected upper extremity more; (4) unilateral loss of hearing (in two animals).

Anatomy.

On this phase of the subject, among the most important studies are those of Kölliker [83], Burdach [26], Edinger [45], Nissl [114], v. Monakow [109], Münzer and Wiener, Ramon y Cajal [28], Bechterew [12], and Probst [129].

We are concerned here, firstly, with the nuclei of the thalamus and their connections with one another; secondly, the thalami-cortical paths; thirdly, the cortici-thalamic; and fourthly, the thalami-spinal paths, if such exist.

Thalamic Nuclei.

The nuclei of the thalamus have been variously classified.

The simplest is that given by Kölliker [83], Obersteiner [117], and Marburg [98]. According to these observers the thalamus is divided into the following nuclei :—

(1) Nucleus anterior or tuberculum anterior.
(2) Nuclei disseminati.
(3) Nucleus medius.
(4) Nucleus lateralis.
(5) Nucleus lateralis ventralis.
(6) Centre median.
(7) Nucleus arcuatus.
(8) Corpus geniculatum externum.
(9) Corpus geniculatum internum.

Nissl [114] describes nineteen nuclei as follows :—

(I.) Anterior nucleus: (1) anterior dorsal; (2) anterior ventral; (*a*) dorso-medial, (*b*) ventro-lateral.

(II.) Anterior median nucleus.

(III.) Median middle nucleus.

(IV.) Ventral nucleus of the "Gitterschichte."

(V.) Lateral nucleus of the "Gitterschichte."

(VI.) Dorsal nucleus of the "Gitterschichte."

(VII.) Nucleus of the median line. (This nucleus is split into a dorsal and ventral part by the median middle nucleus.)

(VIII.) Lateral nucleus (large-celled nucleus).

(IX.) Median posterior nucleus.

(X.) Ventral nucleus: (1) ventral lateral; (2) ventral dorsal; (3) ventral median.

(XI.) Lateral posterior nucleus.

(XII.) Ganglion habenula: (1) lateral; (2) median.

(XIII.) Posterior nucleus.

Von Monakow [107], from a large series of experiments in which he used for the most part Gudden's degeneration method, makes the following classification:—

(I.) Tuberculum anterior: (1) anterior *a*; (2) anterior *b*; (3) anterior *c*.

(II.) Lateral nucleus: (1) lateral *a*; (2) lateral *b*.

(III.) Median nucleus: (1) median *a* (magno cellularis); (2) median *b*; (3) median *c*.

(IV.) Ventral nucleus: (1) ventral *a*; (2) ventral *b*; (3) ventral *c*; (4) ventral anterior.

(V.) Posterior nucleus.

Von Monakow does not, however, consider several points of experimental and pathological import, which must be fully discussed before one can positively connect any one nucleus with a particular part of the cortex. In his investigations large areas of cortex were excised, and the atrophy of the thalamic nuclei was studied by Gudden's method. In excising large areas of cortex there must necessarily be considerable thrombosis of the large arteries, and as a result a part of the blood supply to the thalamus may have been interfered with. The atrophy observed may have resulted in part from this cause. In support of this criticism Beevor, in his recent paper on the blood-supply of the human brain, showed that the same vessels supply the thalamus as supply the superior and middle frontal convolutions.

Furthermore, by destroying a portion of cortex from which fibres arise which only pass through the thalamus, the resulting atrophy of these fibres may produce the appearance of shrinkage of the nuclei without complete disappearance. This, in fact, occurred in some of the nuclei in which changes were described.

Ramon y Cajal [28] uses the following classification of the thalamic nuclei. He conceives the nuclei as divided into three series :—

(I.) Lateral series : (1) external geniculate ; (2) internal geniculate; (3) pulvinar ; (4) radiate nucleus (lattice nucleus of Nissl).

(II.) Intermediate series : (1) angular ; (2) dorsal ; (3) main sensory nucleus (Foco sensitivo), with its accessory or semilunar nuclei ; (4) posterior nucleus.

(III.) Median series : (1) median nucleus ; (2) commissural nuclei ; (3) middle nucleus ; (4) internal nucleus ; (5) ganglion habenula.

Mann, in a preliminary paper, distinguished the nuclei as follows : He adopted primarily the old division into a dorsal and ventral portion. The dorsal he again subdivided into a dorsal and ventral area. The portion bounded by the surface looking towards the middle line he called the ventricular third, the lateral aspect the lateral third. Between these two lies the subventricular third. He worked on the Marmoset monkey, and recognized the following nuclei :—

(I.) Anterior nucleus : No. 1 *a*, No. 1 *b*.
(II.) Ventricular nucleus : No. 2 *a*, No. 2 *b*, No. 2 *c*.
(III.) No. 3 *A*, No. 3 *B*, 3 *B a*, 3 *B b* (3 *B b z*, 3 *B b x*), 3 *B c*.
(IV.) Posterior nucleus : No. 4 *A*, No. 4 *B*.
(V.) Ganglion habenulæ : No. 5 *a*, No. 5 *b*.
(VI.) External geniculate body : No. 6 *a*, No. 6 *b*.
(VII.) Internal geniculate body : No. 7 *a*, No. 7 *b*.
(VIII.) Pineal body.
(IX.) The retinæ.
(X.) Reticular nuclei (these are subnuclei) : No. 9 *a*, No. 9 *b*, No. 9 *c*.
(XI.) Ventral nucleus : zone *a*, zone *b*, zone *c*.

Without further detail it is difficult to compare the nuclei thus designated with those found by other observers.

Recently, while in Professor Obersteiner's laboratory in Vienna, I examined thirty-four mammalian brains (marsupials to man), comparing their optic thalami. This study, together with the brains of the cats and monkeys used in the present investigations, have led me to adopt, at Sir V. Horsley's suggestion, the following classification of the thalamic nuclei until the precise connections of each part with the rest of the brain are determined. It corresponds very closely to the original description by Burdach [26], and at least for the results recorded below seems most practical:—

Nucleus anterior.
Nucleus medius.
Nucleus lateralis (dorsal portion, middle portion, ventral portion).
Nucleus ventralis.
Centre médian.
Nucleus arcuatus.
Pulvinar.

The anterior of these are well seen in fig. 28.

Fibres Between the Intrinsic Thalamic Nuclei.

As to the intrinsic fibres between the thalamic nuclei, I have been unable to find any observations, except the one conjecture by Probst [129] that the nucleus anterior sends its fibres to the caudate nucleus and Lewandowsky's [88] belief that there is a connection between the nucleus anterior and the corpus Luysii.

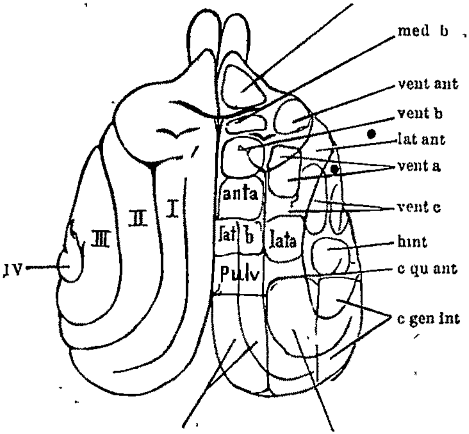

FIG. 1.

This point is discussed under the description of the lesions.

Thalami-cortical Fibres.

Thalami-cortical fibres have been described by Monakow [109], M. and Mme. Dejerine [40], Van Gehuchten [62], Lewandowsky [88], Roussy [135], and Probst [129].

Von Monakow [107], using Gudden's method, allotted certain thalamic nuclei to definite parts of the cortex, and believed that most of the connecting fibres were thalami-cortical. The accompanying drawing (fig. 1) from his article shows these connections. It has been pointed out why the results obtained by this method cannot be accepted as completely satisfactory.

Dejerine [40], in 1901, spoke of thalami-cortical fibres, but did not describe from which part of the thalamus they arose, nor to which part they were distributed. He believed them to be equal in number to the cortici-thalamic fibres.

Van Gehuchten [62], in 1906, said the thalami-cortical fibres had their origin from the lateral nucleus of the thalamus, and that it was supposed that they ended in the central convolutions and paracentral lobule mostly in the post-central convolution and the precentral convolution immediately adjacent.

Lewandowsky [88], in 1907, also spoke of thalami-cortical fibres, but gave no more detailed account of their origin or distribution.

Probst [129], in one case on a monkey, severely injured the capsule near the thalamus, and, as he found degeneration in the cortex, concluded there must be thalami-cortical fibres. He ignored the fact that fibres degenerating from such an extensive lesion may have had some other origin and considered that the accompanying injury to the corpus callosum was not responsible for the degeneration observed in the cortex. Probably some of the fibres were in fact thalami-cortical, but the evidence offered was not adequate.

In Roussy's [135] experiment on the monkey the capsule was not injured, but the injury to the corpus callosum, cingulum, and the slight injury to the superior frontal convolution, might be responsible for some of the degeneration he observed, as he himself suggests. He described numerous fibres passing to the frontal lobes (radiations thalamiques anterieures), and radiations thalamiques moyennes (fronto-parietales), which go to the pre- and post-central gyri and some to the lenticular nucleus.

The frontal fibres he believed came from the nucleus anterior and internal nucleus, while the fronto-parietal fibres came from the "external" (lateral) nucleus. He also found fibres going to the first temporal convolution and to the parieto-occipital region from the posterior part of the thalamus. He observed no connections between the thalamus and subthalamic body. He saw fibres going to the red nucleus, but was not certain whether their condition was genuine degeneration or due to indirect Wallerian degeneration. He observed no fibres going to the cord.

Cortici-thalamic Fibres.

It has been shown by Flechsig [51], Bechterew [12], Edinger [45], Dejerine [40], Kölliker [83], and others, that there probably exist cortici-thalamic fibres. This has been emphatically denied by Lewandowsky [38], and v. Monakow [109] believed they were very few. Van Gehuchten believes they exist. Dejerine, from observations with the Marchi as well as the Weigert method in cases of lesions of the hemispheres in man, described them as going to the external, internal and anterior nuclei of the thalamus, also to the "zone réticulée," pulvinar, ventral part of external nucleus and centre median of Luys.

Thalami-striate. Fibres.

Fibres between the thalamus, corpus striatum, and subthalamic region have also been described, notably by Edinger and Dejerine, but, owing to the difficulties underlying their demonstration, little is known in mammals beyond their mere existence.

Descending Thalami-fugal Fibres.

Investigators are divided into two camps as to descending or thalamifugal connections. One, headed by Bechterew, his pupils, and Wallenberg, believe that there are descending tracts to the cord. The most recent publication on the subject is by one of his pupils, Ernst [47]. This work, being in Russian, has probably not received full recognition.

His conclusions are as follows:—

(1) Degeneration to the medulla occurs from lesions in the posterior part of the thalamus only.

(2) The nucleus anterior and the anterior and middle parts of the thalamus send no descending fibres.

(3) After injury to the posterior and inner part of the thalamus descending degeneration to the external part of the anterior colliculus and to the nucleus ruber occurs.

(4) Two tracts were traced down to the medulla. After injury to Monakow's nuclei med. *a* and vent. *b* one tract runs along the outer side of the fasciculus longitudinalis posterior and ends in the nucleus of the formatio reticularis. The other ends in the nucleus reticularis tegmenti.

Opposed to Bechterew's [8] views are van Gehuchten [62], Dejerine [40], Probst [123], and Roussy [135]. They believe that no descending tracts arise from the thalamus itself.

The explanation of this apparent diversity is due to the lesions employed having been so gross that no accurate conclusion could be drawn as regards the precise source of any descending degeneration. The reader is referred to p. 151, &c., for the facts which fully explain the positive results obtained by Ernst.

DESIGN OF PRESENT RESEARCH.

From the foregoing historical account it will be seen that the fundamental problems requiring solution are:—

(1) The arrangement and relations of the intrinsic nuclei of the thalamus.

(2) The existence, if any, and distribution in the thalamus of cortico-thalamic tracts.

(3) The distribution and origin of fibre-tracts leaving the thalamus.

Without anticipating the detailed summary of results given on p. 180 at the end of this paper, it should be stated at once that the present research has definitely established the correctness of the old view first proposed by Burdach, and for many years the solely accepted mode of regarding the thalamus—namely, that it is essentially composed of three parts: (1) an external lying between the internal capsule and the lamina interna; (2) an internal between the lamina and the median line; (3) an anterior corresponding to the nucleus anterior.

METHODS AND TECHNIQUE.

For the present investigation there were used sixty-one monkeys (*Macacus rhesus*) and thirty-three cats.

The experiments will best be arranged as follows, in three groups. They were all performed by Sir Victor Horsley or myself.

Group I.—Localized lesions of the cortex cerebri and subsequent anatomical investigation of the cortici-thalamic fibres.

Group II.—Localized lesions of the thalamus and subsequent anatomical investigation of the thalami-cortical and other tracts of fibres associated with the lesion.

Group III.—Phenomena observed on electrical excitation of the thalamus and its neighbourhood.

The animals were all anæsthetised with chloroform or ether. The animals in the first two groups were operated upon under strict aseptic precautions, kept alive eighteen to twenty-one days, and then killed with an overdose of ether or chloroform, while those in the third group were killed while still under anæsthesia.

Group I.

The first group consisted of twenty-four cats and thirteen monkeys. Of these a certain number had to be discarded, several because they died of shock and hæmorrhage, several monkeys because they died of diarrhœa before degenerations could be studied, and a considerable number because of indifferent penetration by osmic acid, and a few because the lesions were not in the desired region. There remain to report in this group, therefore, nine cats and ten monkeys. In these eighteen animals very small areas of cortex were destroyed with concentrated nitric acid. The procedure of necrosis is one that has been used before by various observers, by whom, however, alkalies have been usually employed. A small brush with glass bristles on which all but about twenty bristles had been removed was used. This was sterilized and dried, and then dipped into nitric acid. All excess was shaken off and the cortex to be destroyed then painted with the acid. It is well to dry the cortex with small cotton pledgets just before applying the acid, so that it does not spread. As soon as the cortex is whitened, warm saturated bicarbonate of soda solution is poured over it to neutralize any excess of acid. With a little practice extremely small lesions can be made. It is important not to let the acid touch any large vessels, as the ensuing thrombosis produces a larger area of softening. The character of these lesions is shown in fig. 9. The slight depth to which the acid penetrates is shown in the figure. Later several lesions were made with a galvano-cautery, and this method is more satisfactory in that its lesions may be still more accurately defined. This series provided material for study of the cortici-thalamic fibres.

Group II.

The second group contained thirty-five monkeys and nine cats. Several monkeys had to be discarded—four because the lesions were too high and not in the thalamus, two because they died of diarrhœa, one because of poor osmic penetration, and one which became infected; five cats had to be discarded because they died of shock. There remained to study twenty-seven monkeys and four cats. Here the electrolytic method of Sellier and Verger [140] was made use of. In order to produce minute electrolytic lesions at known points in the thalamus, Clarke's stereotaxic instrument was used. This instrument has been recently described in great detail by Horsley and Clarke [75] in their accounts of experiments on the cerebellum. To produce lesions in the thalamus a series of ten animals was first prepared in whom the needle was introduced through the vertex to the frontal region. This was the line of approach used by Probst and Roussy. In our earlier experiments we used the larger needles,[1] measuring 0·5-0·7 mm. in diameter, and sometimes the double needles, measuring 1·5-0·2 mm. in diameter. The lesion produced with the double needles is well shown in fig. 30. Even with the smallest needles, measuring 0·34 mm., there is sufficient injury to the corpus callosum to produce degeneration, which somewhat obscures the picture. It is this fact which probably accounts for the different results obtained by us compared with Probst and Roussy.

For this reason, in the last thirty-one experiments the lesions were made by introducing the needle horizontally through the occipital lobe as shown in fig. 36, where the needle track as well as the lesion is visible.

This produced some degeneration from the occipital lobes, but as the course of these fibres is well known through the work of Monakow [109], Cajal [28], Munk [111], Mott [110], and Probst [126], and can be readily distinguished from lesions in the other parts of the thalamus, it was of no significance. The primary object was to leave the frontal pre- and post-central gyri absolutely intact. As the needle was advanced millimetre by millimetre each point was stimulated with a faradic current, whose strength was regulated by a coil with the Kronecker graduation. These stimulations enable one to know where the needle point is, and, though unipolar, furnished valuable additional data to our stimulation experiments to be described under Group III. However, these unipolar stimulations must be used with considerable reserve for the reasons stated by Horsley and Clarke.

Group III.

In the third group there were thirteen monkeys. In this series the object was to stimulate systematically the thalamus millimetre by millimetre in each lamella to determine what phenomena were obtained at the various points. We have confined ourselves to recording movements of the voluntary musculature as well as movements of the pupil. The visceral phenomena, *i.e.*, blood-pressure changes (recorded by Prus [130] and Ott [119]), changes in temperature (described by

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[1] See Clarke and Horsley [75].

Hale White [68]), and movements of the intestinal tract (described by Bechterew and others), we have not investigated, but on the facts of lachrymation, mastication, and vocalization, however, we have made observations, and believe that these phenomena admit of other explanations.

In these stimulations bipolar electrodes have been used throughout, for with one electrode in the brain and the other at an "indifferent" part of the body, untrustworthy results, due to escape of current, have been obtained. This question of unipolar and bipolar stimulation has been dealt with at length by Horsley and Clarke in their article in *Brain*, 1908. What they found in the case of the cerebellum has been confirmed by the present investigation of the thalamus, for with one electrode in the thalamus and the other on the trunk of the animal facial and neck movements were obtained by the excitation of points in the thalamus which yielded no observable muscular contractions at all when the bipolar needle was used.

POSITION OF THE THALAMIC NUCLEI.

On reference to Horsley and Clarke's paper, it will be found that, for purposes of orientation with Clarke's instrument, the brain is conceived as divided by three planes:—

The sagittal plane, which passes longitudinally between the two hemispheres.

The basal plane, whose four points are determined by the inferior margin of the bony orbits and the centres of the bony auditory meati. The horizontal plane employed for convenience as zero is parallel to this and 10 mm. higher in the average Rhesus monkey.

The frontal or inter-aural plane, perpendicular to the latter, passes through the centres of the two meatuses.

The plane sections of the encephalon, parallel to the sagittal plane, are spoken of as right or left lamella, I, II, III, &c., according to their distance in millimetres from the sagittal (zero) plane.

The height above or below the horizontal plane is designated in letters—thus, A = 1 mm. above or below the plane, B = 2 mm., &c. The numbers 1, 2, 3 designate the distance in millimetres in front of or behind the inter-aural plane.

These planes thus divide the brain into eight segments frontal (right or left) above the horizontal and in front of the inter-aural planes; temporal (right and left) below the horizontal and in front of the inter-aural planes; occipital (right and left) above the horizontal and behind the inter-aural planes; cerebellar (right and left) below the horizontal and behind the inter-aural planes.

The optic thalamus in a normal Macacus rhesus lies entirely in the frontal and temporal segments with the exception of the posterior end of the pulvinar, which lies in the occipital segments.

The thalamic nuclei in a normal Rhesus, according to measurements from a number of our experiments, occupy the following position with reference to this nomenclature.

Nucleus anterior: Lamella II—IV, L—N, 8—12.

Nucleus medius: Lamella I—IV, G—L, 3—8.

Nucleus lateralis: Lamella IV—X, H—L, 4—13.

Nucleus ventralis: Lamella IV—VIII, E—H, 4—10.

Nucleus arcuatus and centre median; Lamella II—V, E—H, 4—6. Pulvinar: Lamella IV—X, anteriorly E—K, two behind inter-aural line to four in front; posteriorly C—G, two behind inter-aural line to four in front.

Results of Cortical Lesions.

On Table I are recorded the results of the cortical lesions and the cortici-thalamic paths thus displayed. From these certain definite conclusions have been reached. The general principle laid down by Monakow that the anterior part of the cortex cerebri is connected with the anterior portion of the thalamus, the middle part with the middle portion of the thalamus, and the occipital region with the posterior part or pulvinar, is in accord with the findings of all workers on this subject, notably Edinger [45], Cajal [28], Kölliker [83], van Gehuchten [62], Ferrier and Turner [49], and others.

Zuckerkandl, Edinger [45], Kölliker [83], and Dejerine [40] proved that the nucleus anterior belonged to the olfactory system, and Edinger [45], Hitzig [72], Munk [111], Ferrier and Turner [49], Probst [129] and others showed the intimate connection between the occipital lobe and the pulvinar.

It is with the middle portion that the present work is most concerned, and we have examined the conditions as they exist in the cat as a type of the Carnivora and in the Macacque monkey Macacus rhesus. As regards the cat, the relation of the thalamus to the gyrus pre- and post-crucialis, gyrus sigmoidalis, gyrus lateralis, and gyrus suprasylvius medius (cat), the Marchi method has yielded different results from those obtained by Monakow, who employed Gudden's method.

The fibres that actually end in the thalamus and arise from these regions all enter the nucleus lateralis (lateralis *a* and *b* of Monakow) or nucleus ventralis (ventralis *a*, ventralis anterior and ventralis *c* of Monakow). The various parts of the cortex cannot be said to be restrictedly connected with one or the other of these nuclei, though in every instance where the pre- and post-crucial gyri were involved the fibres passed to the nucleus lateralis and ventralis. In no instance have any fibres been seen going to the median nucleus or centre median. The lamina interna constitutes an absolute dividing line as regards the distribution of these fibres. This structure thus forms a most important line of demarcation both for the cortici-thalamic as well as thalami-cortical fibres, as will appear later (*see* fig. 2 *et seq.* Cat 99).

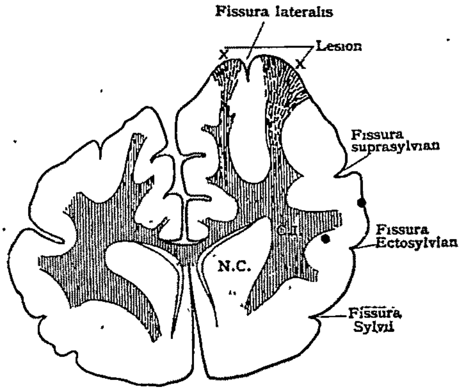

FIG. 2.—Cat 99 (I).

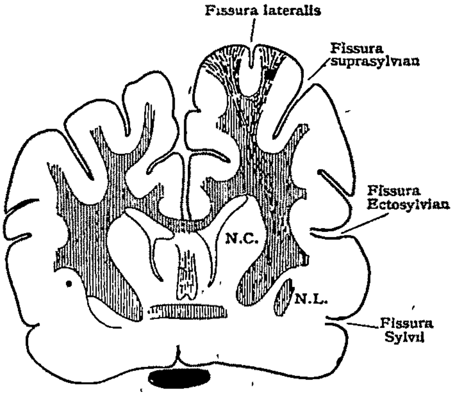

FIG. 3.—Cat 99 (II).

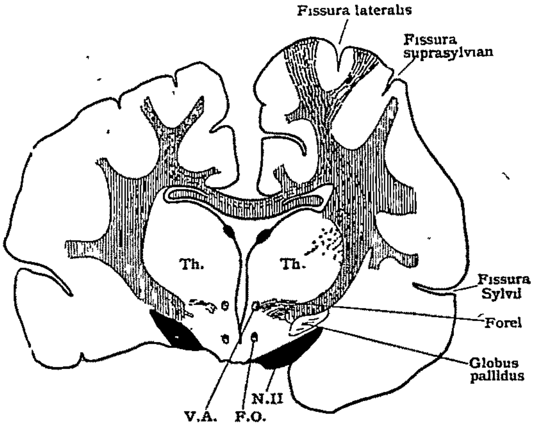

FIG. 4.—Cat 99 (III).

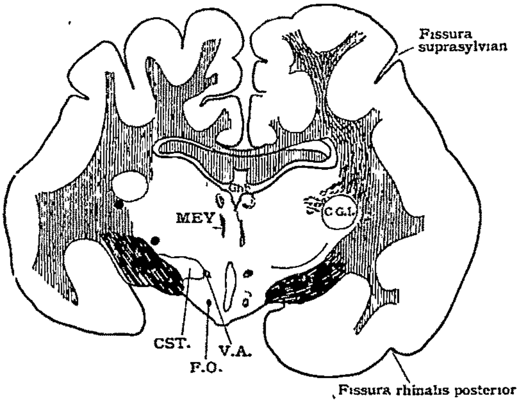

FIG. 5.—Cat 99 (IV).

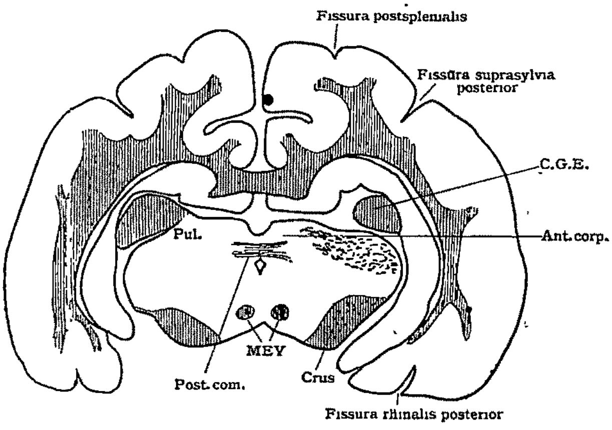

FIG. 6.—Cat 99 (V).

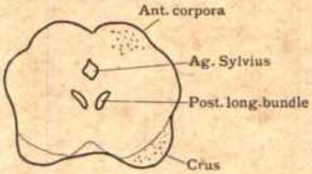

FIG. 7.—Cat 99 (VI).

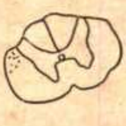

FIG. 8.—Cat 99 (VII).

Besides the fibres ending in the thalamus there are also numerous pallio-tectal fibres, as described by Edinger, Beevor and Horsley. It is this group which must be distinguished carefully from the cortici-thalamic fibres.

On this point several of the animals throw some light, *i.e.*, whether pallio-tectal fibres come in larger numbers from one part of the cortex than another. In Cats 127, 131 and 283 the lesion was confined entirely or for the most part to the so-called motor area—*i.e.*, gyrus pre- and post-crucialis[1]—and gave rise to no pallio-tectal fibres, or at most extremely few, while from lesions in the suprasylvian or ectosylvian

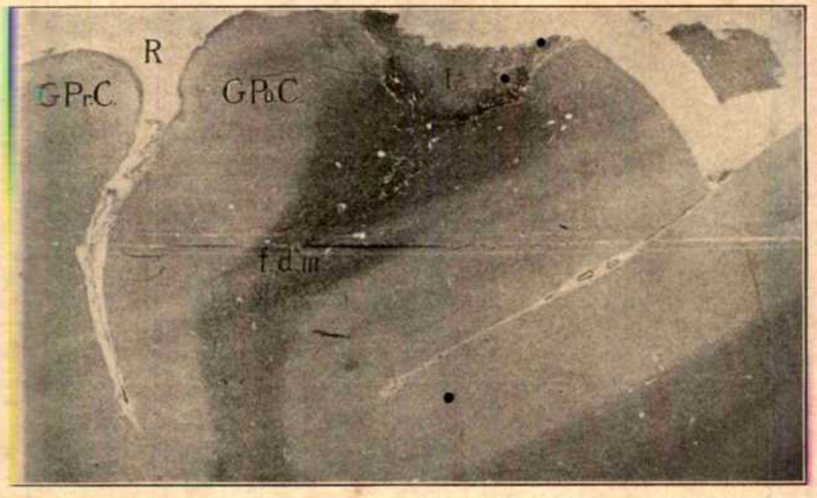

FIG. 9.

Macacus rhesus. Marchi preparation, showing restricted character of the lesions employed in Table I.

R = Fissura centralis.
G.Po.C. = Gyrus postcentralis.
G.Pr.C. = Gyrus precentralis.
l. = lesion.
f.d.m. = degenerated pyramidal fibres.

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[1] In the excellent text-book by Flatau and Jacobson [50] these gyri are spoken of as pre- and post-central. This attempt to correlate these gyri with the gyri of the same name in the human and anthropoid cortex is not satisfactorily proven. I therefore adhere to the old nomenclature.

regions many such fibres arise. The meaning of this fact seems perfectly evident, for from the pre- and post-crucial gyri no ocular or auditory stimuli can be elicited, while from the ectosylvian and suprasylvian gyri movements of eyes and ears are obtained upon faradic stimulations.

In the monkey (*Macacus rhesus*) only the pre-central and post-central gyri (*see* figs. 10 *et seq* and Table I) were explored by limited lesions with a view to determine their relation with the thalamus, and what difference, if any, existed between the thalamic fibres arising from the leg and arm area. In the monkey, as in the cat, the cortici-thalamic fibres were traced exclusively to the nucleus lateralis and nucleus lateralis (ventral part). No fibres passed beyond the lamina interna (*see* figs. 10 *et seq*)

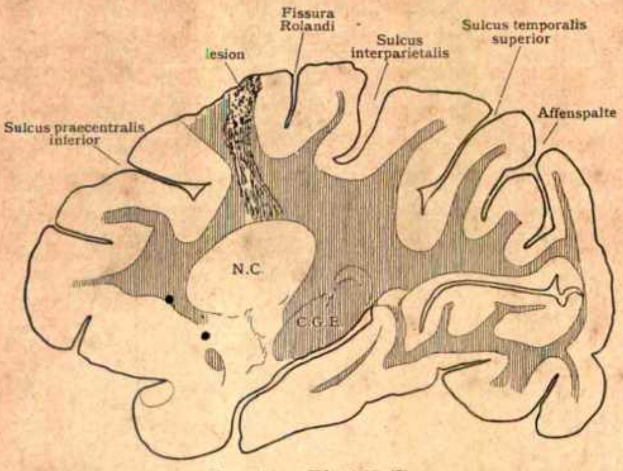


FIG. 10.—Rh. 143 (I).

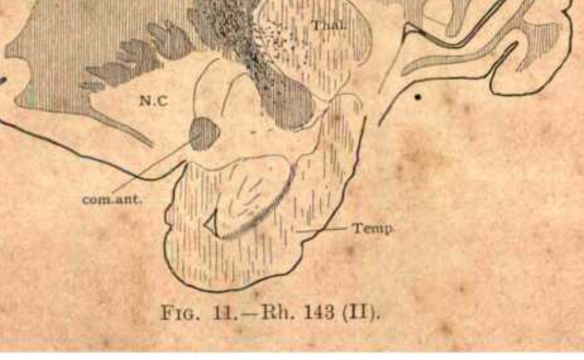


FIG. 11.—Rh. 143 (II).

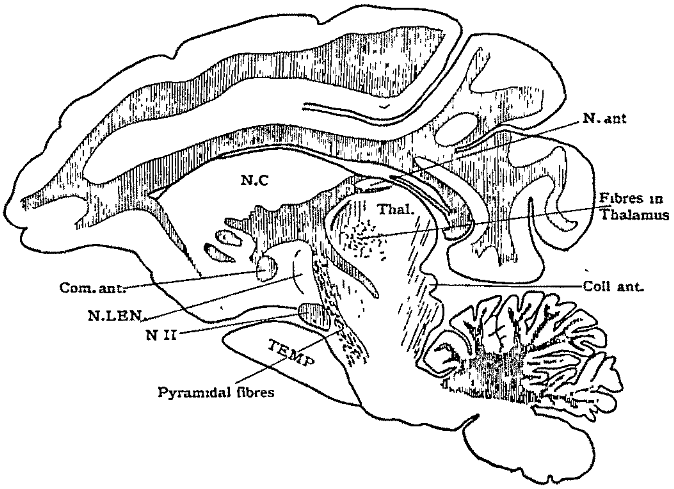


FIG. 12.—Rh. 143 (III).

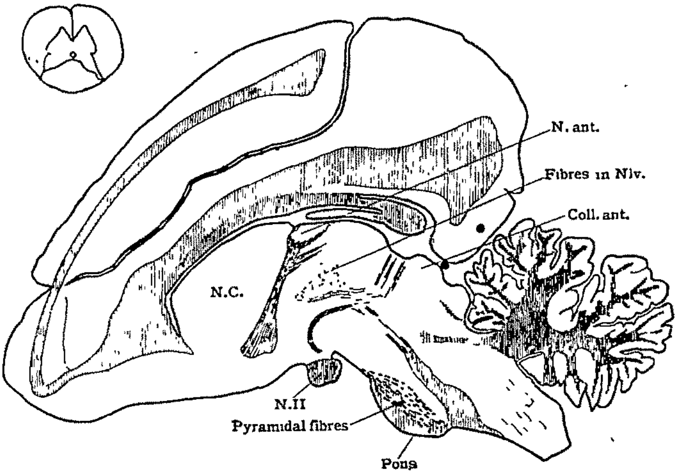


FIG. 13.—Rh. 143 (IV and V).

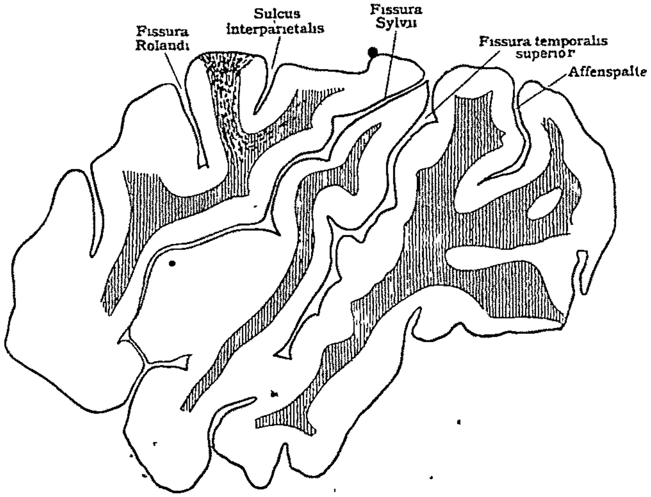


FIG. 14.—Rh. 165 (I).

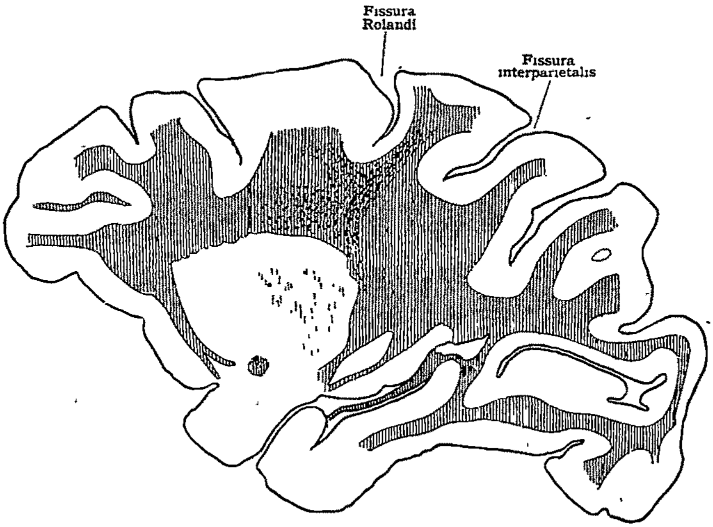


FIG. 15.—Rh. 165 (II).

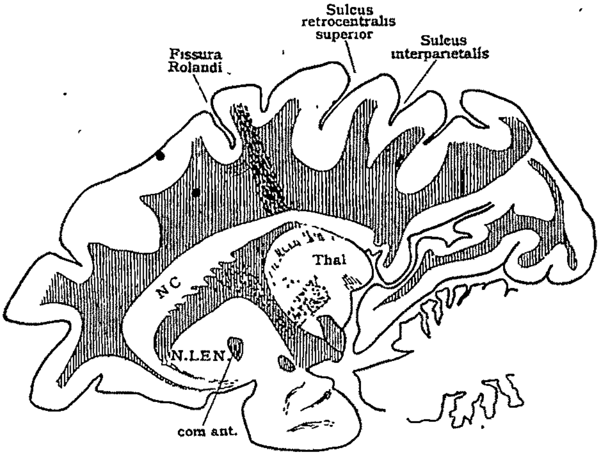


FIG. 16.—Rh. 165 (III).

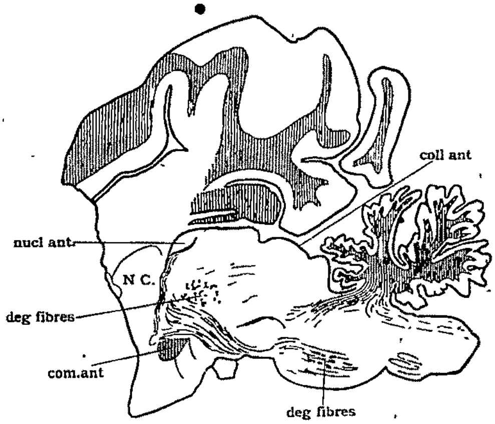


FIG. 17.—Rh. 165 (IV).

for the distribution of fibres from each of these gyri). In view of the controversy as to the functions of these two gyri the quantitative relation between the fibres going to the thalamus seemed of particular interest, as possibly throwing some light on this question. It must, of course, be remembered that in the Macacque brain the pre- and post-central gyri are not as definitely differentiated as in the anthropoids or man, yet the movements on cortical stimulation are all much more distinct pre-centrally By destroying the pre-central arm or leg areas we have endeavoured to find out whether these movements had also post-central localization, or were only due to spread of the current As far as the thalamic distribution of cortici-thalamic fibres could be made out there seems to be no difference between these two gyri. No essential difference was observed between the distribution of the fibres from the leg and arm areas, but before an absolute opinion can be rendered on this point far more work is necessary than it has been possible to do here.

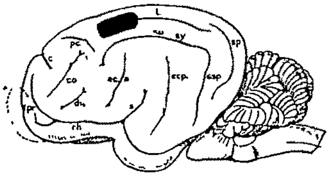
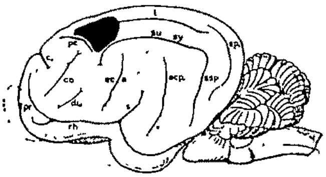
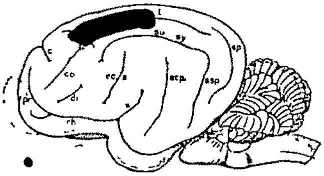
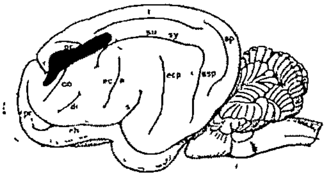
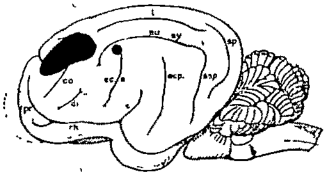
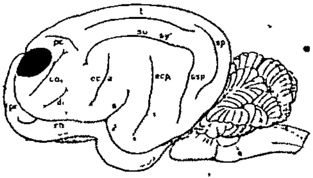
Pallio-tectal System.

The question of the structural changes set up by lesions in the thalamus has so far been discussed without reference to fibres which are transmitted through the thalamus, such as the system of fibres which directly connects the pallium with the tectum or corpora quadrigemina. These fibres, first observed by Edinger [43] and Boyce [24], and subsequently described in the higher mammals by Beevor and Horsley [20] and Simpson [143A], consist of two sets, dorsal and ventral. The dorsal group leave the internal capsule opposite the upper third of the thalamus and run backwards to gain the outer side of the colliculus anterior, whereas the ventral group do not take origin from the pyramidal system until the crus is reached, when numerous fibres cross the corpus Luysii and extend to the posterior colliculus

The present research has fully confirmed this grouping of the pallio-tectal system, and has further extended knowledge of the regions of the cortex whence the palliol-tectal fibres arise, so far as the temporal lobe and Rolandic regions are concerned.

The lesions of the temporal cortex given in Table I produced a characteristic degeneration of the pallio-tectal system, fibres being distributed to both colliculi as well as to the thalamus and pons, as described by Beevor and Horsley [20] (*see* figs. 18 *et seq.*, Cat 103).

TABLE I.—*Cortical Lesions.* (A) *Cat.*

No.	Lesion	Diagram	Fibres to homo lateral thalamus
Cat 99	Area 10 × 5 mm. on both sides of sulcus lateralis, anterior third of upper border of gyrus suprasylvius		Fibres in two groups to lateral nucleus separated by external geniculate; fibres dorsal to this mostly pallio-tectal, though some few break up in lateral nucleus of thalamus and pulvinar; those ventral to geniculate all end in lateral nucleus (ventral part)
Cat 135	Area 5 × 11 mm. Anterior half of gyrus anterolateralis and anterior end of gyrus suprasylvius medius		Fibres running through posterior part of thalamus; a few fine fibres also break up in nucleus lateralis (ventral portion)
Cat 139	Area 5 × 15 mm. Posterior part of gyrus post-cruciatus; gyrus suprasylvius medius and a corner of gyrus anterolateralis		A few fibres to nucleus lateralis ventral portion
Cat 127	Area 4 × 15 mm. Gyrus cruciatus posterior and anterior; gyrus suprasylvius (anterior extremity) coronalis (slightly)		A few very fine fibres in ventral portion of lateral nucleus
Cat 283	Area 8 × 12 mm. Post-crucial gyrus, anterior end of gyrus anterolateralis and gyrus suprasylvius medius		Fibres to ventral part of nucleus lateralis
Cat 131	Area 5 × 10 mm. Gyrus cruciatus anterior and posterior		Fine degeneration in nucleus lateralis ventralis

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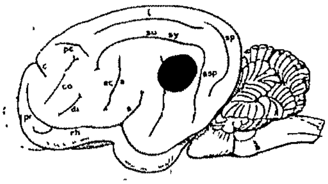
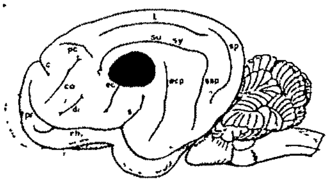
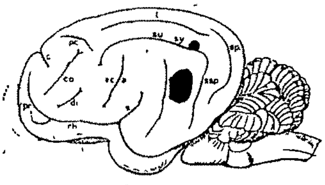
[1] These figures refer to the strength of the

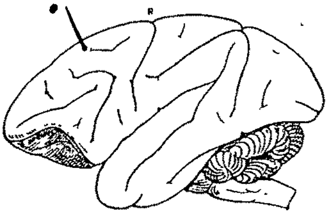
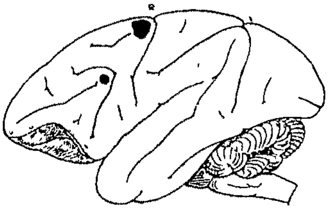
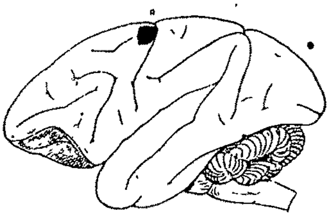
(The number refers to the page in the Notes Ledger.)

Fibres to other regions	Symptoms	Remarks
Fibres pass into middle third of crus; from here behind corpus subthalamicum they pass up into anterior colliculus. A few fine fibres go anteriorly into subependymal tissue (fasciculus subcallosus), these traced forward to back of rostrum, and posteriorly only a short distance opposite about the middle of thalamus Cord no degeneration	Slight loss of sense of position of contralateral fore-leg. No tactile and temperature sensory changes. Grips floor poorly with contralateral fore-leg	Electrical stimulation of sigmoid gyrus on operated side showed motor cortex normally active. Drooping and pricking of ear obtained from upper end of gyrus ectosylvius anterior. The latter weak Current 2000[1].
Pallio-tectal fibres in large numbers running across posterior part of thalamus to anterior colliculus; a very few degenerated fibres in pyramidal tract. Subependymal fibres are half the size of pyramidal fibres and take their origin to the inner side of the pyramidal fibres	Right fore-leg at times seems a little weak in gripping the floor. All else negative	
A few fibres to nucleus lateralis of contralateral thalamus. Fibres to nucleus of crus and tuber cinereum of both sides. Pallio-tectal fibres running through lateral nucleus and up in large numbers through nucleus ventralis and locus niger. These interlace with the fillet fibres. Many of large pyramidal fibres end in pons Cord clear	Slight loss of sense of position of right fore-leg. All else negative. Grips floor poorly	
The degenerated fibres occupy the inner and middle third of the crus. Cross pyramidal tract degenerated in cord. Subependymal tissue shows no degeneration. A very few pallio-tectal fibres belonging to the cruro-tectal group	Loss of sense of position of right fore and hind leg Loss to pain and temperature on right extremities. Grips floor poorly with right extremities	
A few inferior crural pallio-tectal fibres pyramidal degeneration in cord	Loss of sense of position of right fore and hind leg; grips floor poorly with both these legs. Cold water not felt on both contralateral legs	
Extensive degeneration in pyramidal tract in cord	Slight loss of sense of position in right fore-leg, grips floor weakly with right fore-leg. Tactile sensation normal	Stimulation of the gyrus sigmoidalis gave strong contraction of right hind leg, but very weak ones of right fore-leg.

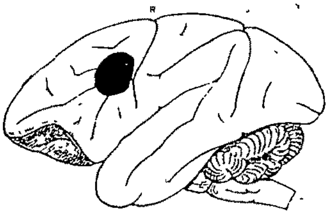
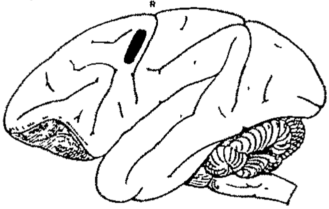
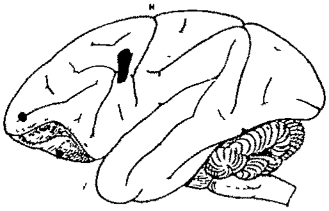
Faradic current as indicated on the Kronecker coil.

No.	Lesion	Diagram	Fibres to homo lateral thalamus
Cat 151	Area 10 × 10 mm. Gyrus ectosylvius posterior and medius	 l su sy sp pc c co ec s ssp pr di rh	Fibres enter posterior part of nucleus lateralis above and a few below external geniculate; some also go into pulvinar
Cat 181	Area 7 × 13 mm. Gyrus ectosylvius anterior and medius; top of gyri Sylvii anterior and posterior	 l su sy sp pc c co ec ecp ssp s pr di rh	Fibres to ventral and *posterior* portion of nucleus lateralis
Cat 103	Area 5 × 5 mm in gyrus ectosylvius posterior. A small injury in the gyrus suprasylvius medius	 l su sy sp pc c co ec s ssp pr di rh	Fibres run through posterior part of lateral nucleus to anterior colliculus: some break up in lateral nucleus and in pulvinar region

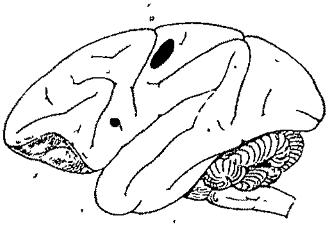
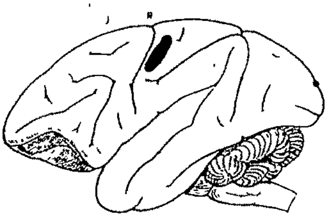
(B) *Monkey* (*Macacus*

Rh. 189	Leg area paracentral lobule, corona radiata (needle track)		Fibres break up in nucleus lateralis and zone grillagé
Rh. 317	Precentral gyrus leg area		Fibres (collaterals of second degree) to second, third, and fourth, fifths of lateral nucleus and to nucleus of lattice layer
Rh. 327	Precentral gyrus leg area		Fine collaterals to middle third of nucleus of lattice layer and lateral nucleus

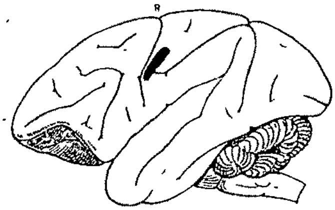
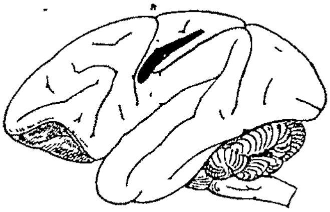
Fibres to other regions	Symptoms	Remarks
Pallio-tectal fibres to anterior colliculus and a few to posterior colliculus, Türck's bundle ending in pons as low as most anterior plane of fourth nucleus; fibres in stratum zonale pass into anterior colliculus; some fine fibres to internal geniculate. Cord no degeneration	Nil.	
Pallio-tectal fibres. Fibres in Turck's bundle	Nil.	
Fibres in capsule pass down to crus in outer third, including Turck's bundle. The internal portion except Turck's bundle run up into anterior colliculus and posterior colliculus. Turck's bundle ends in upper part of pons. Subependymal fibres run forward and back a very short distance. Cord no degeneration	Responds normally to all tests. No changes either motor or sensory	Gyrus sigmoidalis responds normally to faradic excitation.
Rhesus)—(1) *Gyrus Precentralis.*		
Nil	Right arm and leg paretic	This case is also reported in Table II under Thalamic Lesions.
Dorsal third of corpus callosum to symmetrical point in opposite gyrus precentralis	(See later communication)	No pallio-tectal fibres.
Few fibres scattered through the vertical depth of the corpus callosum, few fibres scattered over crossed pyramidal area in cord	(See later communication)	Lesion very superficial; very few Betz cells affected.

No.	Lesion	Diagram	Fibres to homo-lateral thalamus
Rh. 185	Precentral gyrus; arm area almost completely destroyed		Fine fibres occupying middle third of lateral nucleus vertically and antero-posteriorly
Rh. 143	Precentral gyrus arm centre, and lower part of leg centre		Fine fibres run to ventral part of nucleus lateralis
Rh. 301	Precentral gyrus arm area		Fibres to second and third fifths of the nucleus of the lattice layer and the lateral nucleus

(2) *Gyrus*

Rh. 177	Postcentral gyrus opposite arm and leg centre; thrombus in corona radiata destroying presumably also precentral gyrus fibres		Fine fibres enter middle third of lateral nucleus and break up in its outer portion, occupying in area a narrow vertical slip
Rh. 165	Postcentral gyrus opposite arm and leg centres		Fine fibres to nucleus lateralis and to its ventral portion

Fibres to other regions	Symptoms	Remarks
Pyramidal degeneration in the cord	Paresis of right arm.	
Pyramidal fibres in cord	Two days after operation hemiplegic attitude of right arm and leg, knee-jerks equal; no extensor response of big toe Eighteen days later: knee-jerk more active right than left, hemiplegic attitude of right arm, dorsal extension of toes of right foot; right big toe flexor, left toes all flexor, clumsy in jumping; sensation apparently normal (See later communication.)	Electrical stimulation gave with coil 500, movements of right arm, face and eyes; no movements of right leg; thumb movements obtained pre- and post-centrally.
Arcuate fibres pass to upper third of gyrus precentralis; generally distributed fibres in corpus callosum pass to opposite gyrus precentralis		
Postcentralis.		
Fibres in pyramidal tract	Uncertain with right arm and leg when jumping; left arm used more readily than right; weak in right arm and leg; no alteration of sensation to be made out	Arm, leg and face centre excitable with faradic stimulus.
Nil	No motor or sensory changes to be made out, knee-jerks equal on both sides	Cortical stimulation showed no definite results, as monkey weak from diarrhœa, killed on eighteenth day.

No.	Lesion	Diagram	Fibres to homo-lateral thalamus
Rh. 300	Postcentral gyrus opposite arm centre		Fine fibres to ventral half of nucleus of lattice layer and ventral half of nucleus lateralis
Rh. 299	Postcentral gyrus opposite arm centre		Fine collaterals to nucleus of lattice layer and corresponding ventral half of the lateral nucleus

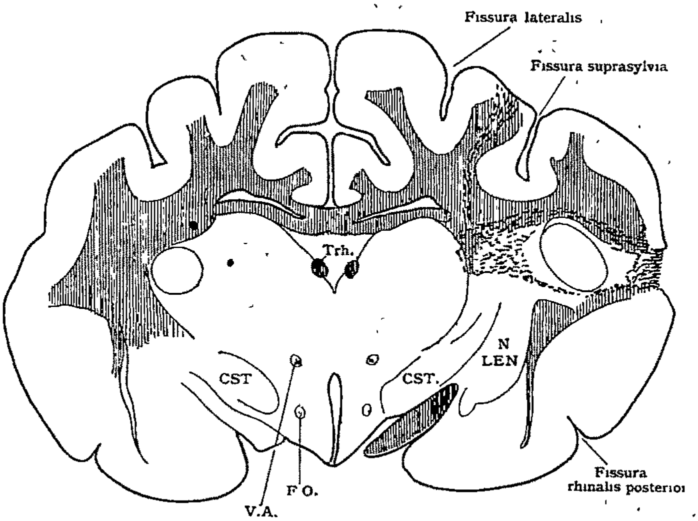


Fig. 18.—Cat 103 (I).

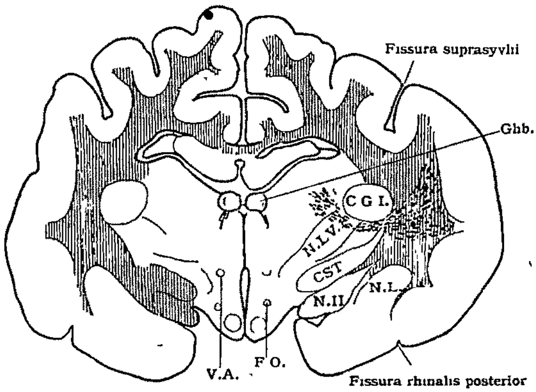


Fig. 19.—Cat 103 (II).

Fibres to other regions	Symptoms	Remarks
Medium fibres in corpus callosum crossing to corresponding contralateral cortex; very few fine collaterals to contralateral thalamus	(See later communication)	No pallio-tectal fibres; no pyramidal fibres traceable beyond pons.
Numerous fibres crossing corpus callosum in dorsal half to corresponding contralateral cortex; arcuates to upper part of postcentral gyrus from lesion; fine collaterals to corresponding contralateral thalamus nuclei	(See later communication)	No pallio-tectal fibres; pyramidal fibres of two classes—(1) the majority fine, (2) a few coarse. The former not traceable beyond the pons. The coarser extend in the cord as low as the lumbar region.

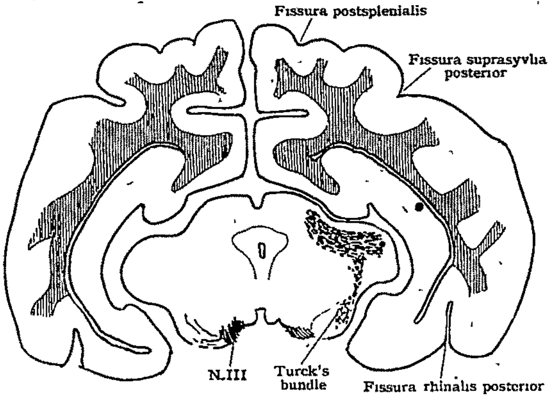


FIG. 20.—Cat 103 (III).

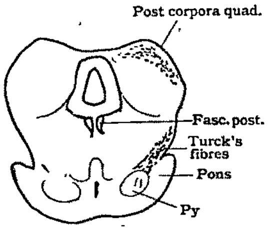


FIG. 21.—Cat 103 (IV).

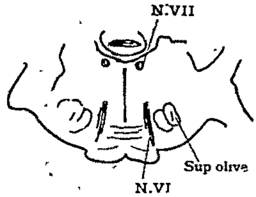


FIG. 22.—Cat 103 (V).

The lesions of the Rolandic region (excito-motor area), shown in Table I, are particularly interesting, as they appear to bring out a point which, if correct, would be of both morphological and physiological importance.

In the present research the limited lesions of the gyrus precentralis in the monkey (five operations), and of the sigmoid gyrus in the cat (one operation), correspond to the findings of Beevor and Horsley and of Simpson respecting the post-crucial gyrus of the dog, in that they were not followed by any pallio-tectal degeneration. On the other hand, those lesions in the cat (see Table I) in which the gyrus coronalis was involved pallio-tectal fibres were found, and from a re-examination by Sir Victor Horsley of the series made by him in 1900-1902, by the undermining method, it is evident that in those experiments the corona radiata fibres of the gyrus coronalis were injured, and consequently that the pallio-tectal degeneration in the cat, observed by Beevor and Horsley, referred to lesions which involved the entire excito-motor cortex and were not limited to the sigmoid gyrus alone.

The interpretation to be put upon these facts is that while the cortical excito-motor area for the conjugate deviation of the head and eyes may be associated with the colliculi by pallio-tectal fibres, that for the limbs is not (*see* figs. 23 *et seq.*).

Further investigations on the monkey will decide this point.

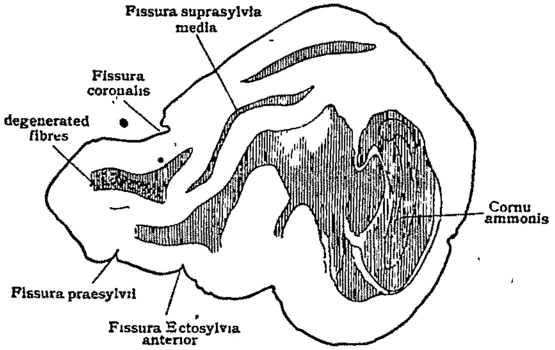


FIG. 23.—Cat 131 (I).

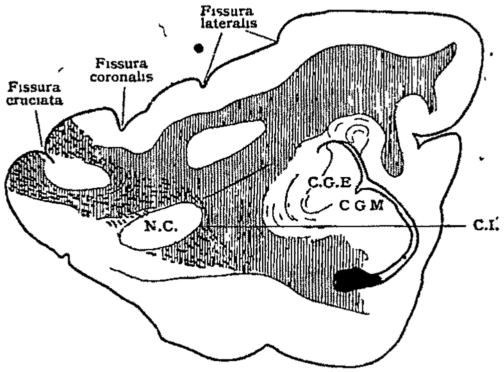


FIG. 24.—Cat 131 (II).

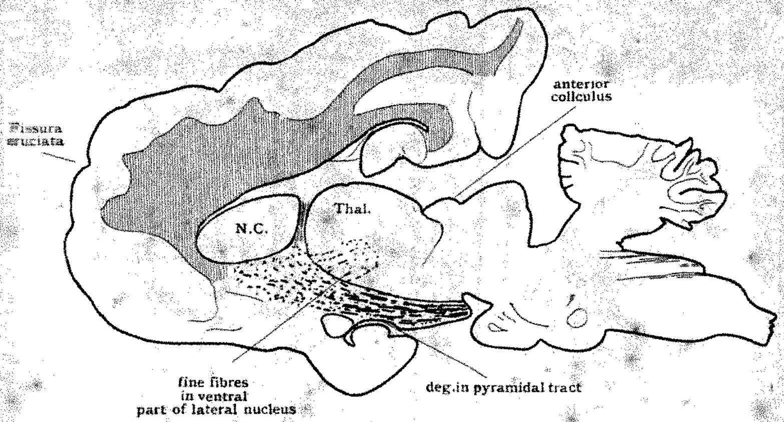


FIG. 25.

ELECTROLYTIC LESIONS IN THE THALAMUS.

Table II contains a description of the various thalamic lesions made in this research with the degenerations arising from each. Figs. 26 *et seq* are microphotographs of the lesions. In this way the need of lengthy descriptions of the lesions is met, and the matter made clearer. Each nucleus will be considered in detail, its relation to the rest of the thalamus and its connections with the rest of the nervous system defined, as far as is shown by these experiments. No tracts will be discussed other than those affected by the lesions.

Nucleus Anterior. (Figs. 26 and 27.)

This nucleus sends many fibres of very fine calibre along the ventral side of the caudate nucleus near its middle and anterior end. By the time the lateral side of the caudate is reached, practically all these fibres have entered the caudate. The few remaining fibres enter the caudate on the external, lateral or capsular side. No fibres pass to the cortex, and this fact further tends to substantiate Edinger's view that the nucleus caudatus belongs to the rhinencephalon. As the fibres from the anterior nucleus lose their medullary sheath as soon as they have entered the caudate, it is impossible to determine by this method how much of, or which part of, the caudate is specially connected with the anterior nucleus. The only fibres that pass back posteriorly are very fine ones, moderate in number, which lie first in the lamina interna and then break up in the outer part of the median nucleus.

I.—LESIONS OF NUCLEUS ANTERIOR.

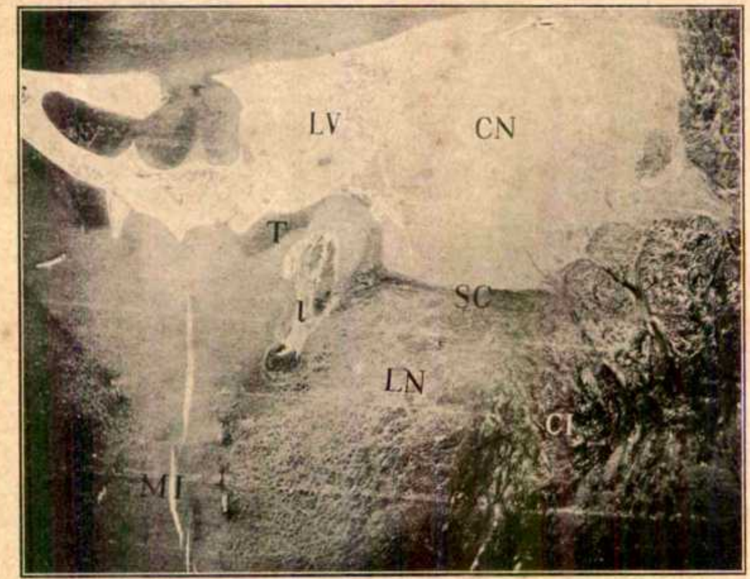


FIG. 26.—[Rh. 189.]

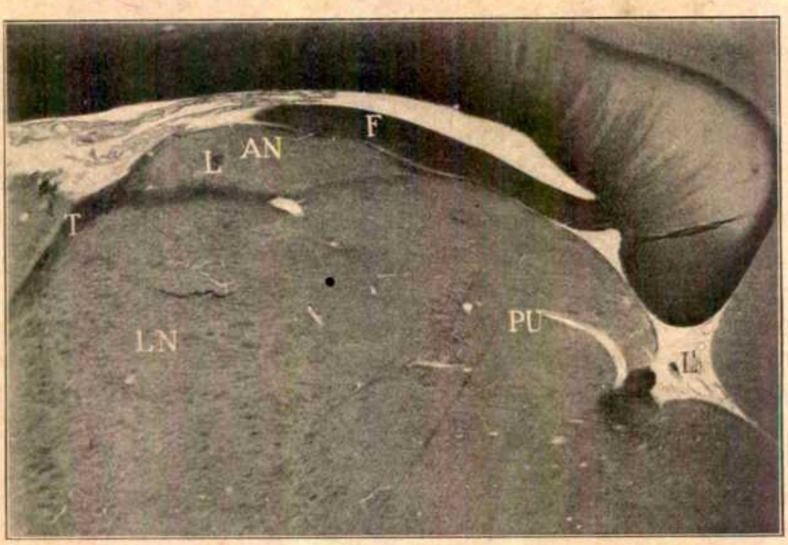


FIG. 27.—[Rh. 222.]

L by AN = The localized lesion in the nucleus anterior. A thrombosed vessel is marked L also just below the splenium.

No fibres were observed passing to any other region. The other thalamic nuclei were all clear. There were no degenerated fibres seen in the lower spinal axis, except in the pyramidal tract, in those cases in which the motor cortex was injured by the passage of the needle. It would seem, therefore, from the present research that the anterior nucleus is an intermediate station between the corpora mammillaria, from which it receives impulses coming through Vicq d'Azyr's bundle and the caudate nucleus.

As seen in fig. 26 S C, the fibres which, as above described, pass out to gain the caudate nucleus are gathered together in a felt-work or capsule separating the nucleus caudatus from the neighbouring more ventral nucleus lateralis. This is evidently a prolongation outwards of the stratum zonale, and, as it probably contains several systems of fibres, it should receive special attention and designation, such as *stratum subcaudatum.*

Nucleus Medius. (Figs. 28-30, inclusive.)

In Rhesus 169, 288, and Cat 95, lesions were made in the median nucleus. The lesions in the two monkeys were sufficiently far from the lamina interna to leave it absolutely intact. They constitute, therefore, a group by themselves, for which Cat 95 but furnished confirmatory evidence, since in that experiment the lesion involved slightly the internal superior third of the lateral nucleus.

The two pure lesions present a picture resembling in most respects that seen in the nucleus anterior, and lead to the inevitable conclusion that the latter must be classed in the same category with the median nucleus as part of the internal thalamic segment (Burdach).

Group I.—Lateral Fibres.

The outwardly directed fibres which become visible after lesions of the median nucleus pass out into the lamina interna at its superior portion, just below the nucleus anterior, and run obliquely across the superior part of the lateral nucleus, and end in the lower border of the caudate nucleus, where they break up into a large number of smaller fibres. In passing through the superior part of the lateral nucleus the fibres of the median are strictly confined to a triangular area bounded dorsally by the caudate nucleus, externally by the remainder of the dorsal third of the lateral nucleus, and internally by the anterior nucleus and lamina interna. As more fibres seem to enter this triangular area

II.—Lesions of Nucleus Medius.

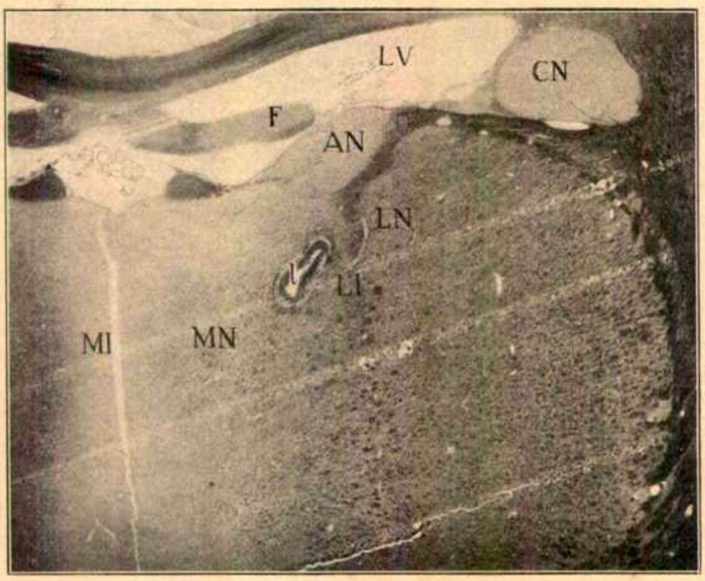


Fig. 28.—[Rb. 228.]

Lesion confined to outer segment of nucleus medius and sending fibres into lamina interna, &c.

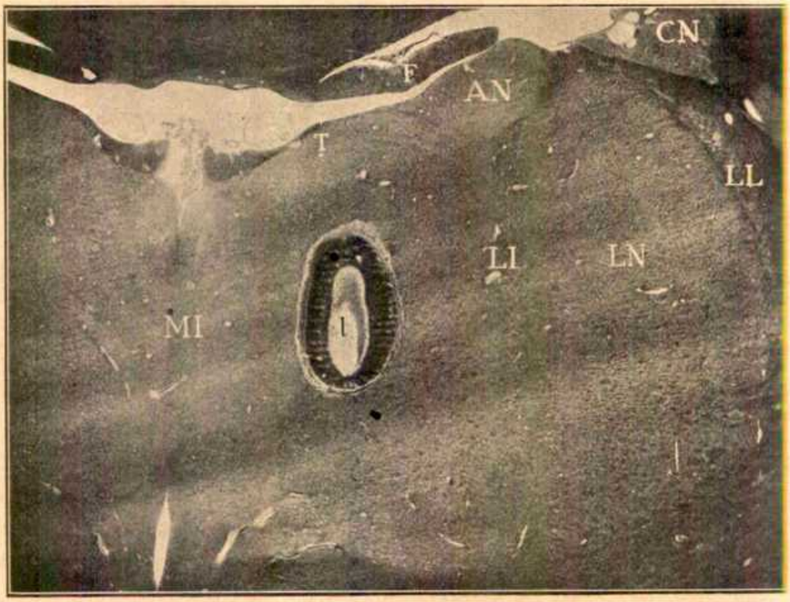


Fig. 29.

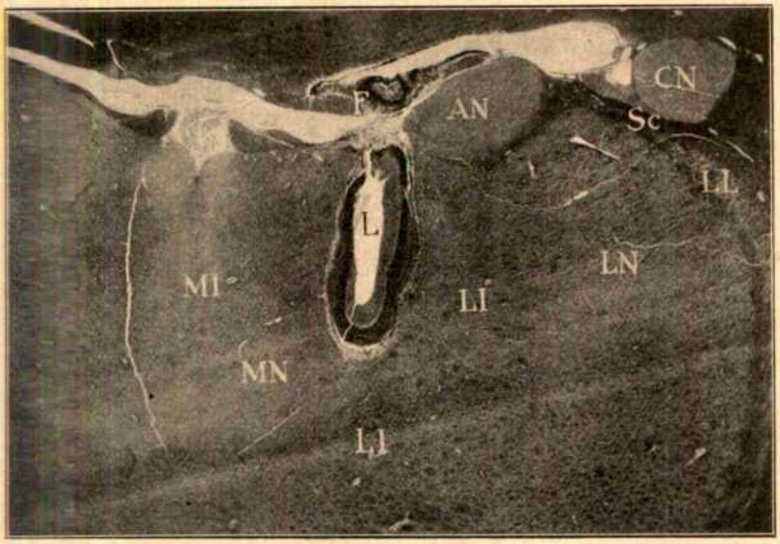


FIG. 30.—[Rh. 119.]

Lesion by vertical penetration. Numerous fibres passing out to stratum subcaudatum, S c. Note track of needle and electrolysis of fornix, F.

than pass beneath the nucleus caudatus, some of the fibres presumably end in the dorsal third of the lateral nucleus.

Group II.—Median Fibres.

The fibres proceeding inwards from the lesion are far fewer in number, they have an oblique inward course, are of fine calibre, and run but a short distance. Their direction is towards the lower and posterior part of the commissura mollis, but they cannot definitely be traced beyond about 2 mm. in the median nucleus. Their direction, however, suggests that their destination is the point mentioned above, which corresponds to the position of the nucleus reuniens first described by Edinger [45] in lower vertebrates, observed by Ziehen [161] in the marsupials, and which I [137] found to be constantly present in the mammalian thalamus.

In Rh. 169 a few fibres were traced to the centre median.

The foregoing were the only degenerations observed after lesion of the median nucleus.

Nucleus Lateralis. (Figs. 28-38 inclusive.)

If the nucleus lateralis be divided into thirds—dorsal, middle and ventral—it is easier to present what has so far been absolutely ascertained by preceding authors concerning the anatomical relation of the nucleus.

The only points which may be properly termed proved are (1) the termination of the fillet, and (2) the ending of the superior peduncle of the cerebellum. Of these the former, first established by Monakow [109] and by Mott [110], and subsequently confirmed by all workers, has been further firmly established by Ramon y Cajal's description of the "nucleus" in the middle and ventral third of the nucleus lateralis, which he termed the "foco sensitivo" and its accessory nuclei.

The erroneous views of Tschermak [151], unfortunately accepted by many writers, viz., that the fillet fibres pass through the thalamus to the post-central region of the cerebral cortex, need not be now discussed; but the question of genuine transmission fibres running in this part of the thalamus becomes of great importance, as many—*e.g.*, the pallio-tectal series—pass through the lateral nucleus.

In three experiments in the present investigation the distribution of the fillet fibres as described, viz., in the ventral third and lower half of the middle third of the lateral nucleus, was well shown.

As regards the second point,[1] the yet unpublished results of a large series of experiments by Horsley and Clarke show that an important part of the distribution of fibres of the superior peduncles occurs in the upper part of the nucleus lateralis just above that of the fillet. This is in accord with the results of the stimulation experiments described below. At the same time it will be remembered that the rest of the distribution of the peduncle in the thalamus is wide—*i.e.*, in the ventral nucleus (Probst [123A]) and in the inner segment of Burdach of both thalami (van Gehuchten [62]).

The details of the cases of lesions strictly confined to the nucleus lateralis must now be considered, and particularly from the point of view of their demonstrating the existence and localization of the system of thalami-cortical fibres, which, though often described with confidence, had, as Roussy [135] shows, never been proved to exist by a pure lesion of the thalamus and consequent degeneration, until his experiment on a monkey.

The pure lesions of the nucleus lateralis obtained in the present research are seven in number, viz., six monkeys and one cat.

They are distributed as follows: Dorsal third, 228, 232, 240; middle third, 261, 270, 247 (cat); ventral third, 272 (dorsal border).

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[1] This whole question, and especially the bilateral distribution of the peduncle, will shortly be discussed by Horsley and Clarke.

As before said, each lesion produces, as a rule, an external or laterally running group of degenerated fibres and an inwards directed or mesial group.

III.—Lesions of Nucleus Lateralis.

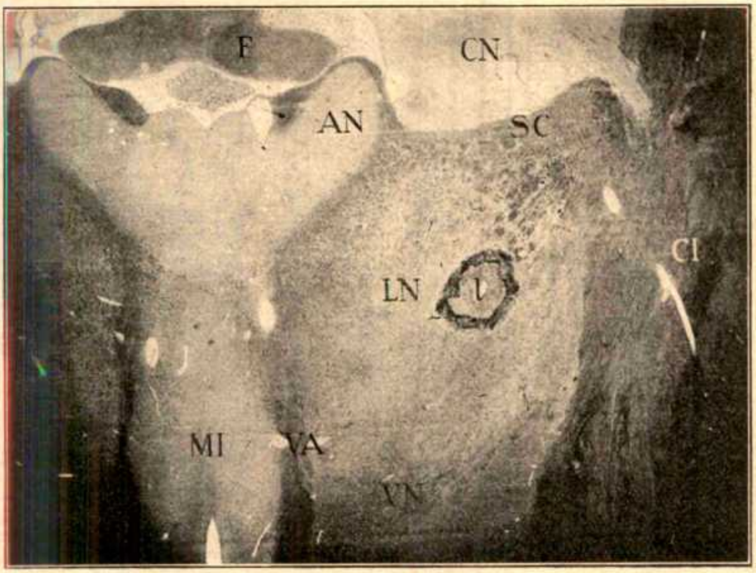


Fig. 31.

Lesion of upper third of nucleus lateralis, showing strong bundles of degenerated fibres running up and out into capsula interna and stratum subcaudatum.

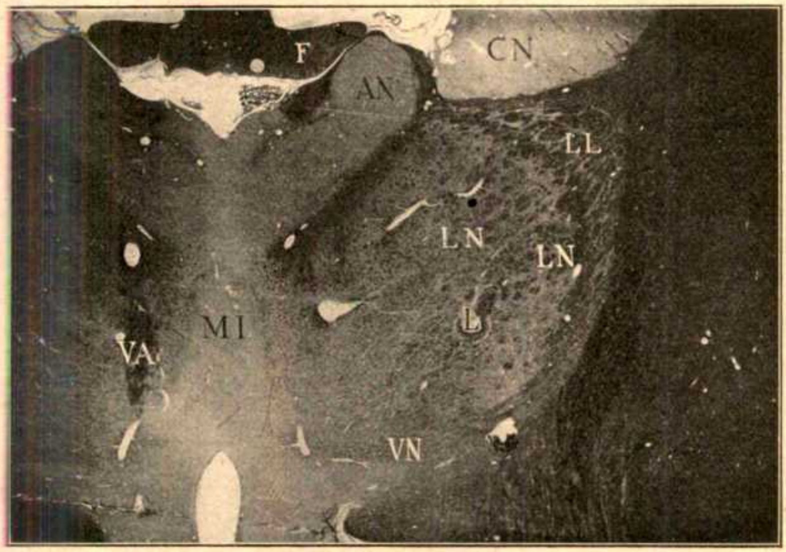


Fig. 32.—[Rh. 261.]

Extremely localised circular lesion in nucleus lateralis.

Group I.—Lateral Fibres.

The first striking fact is, then, whereas from the median and anterior nuclei no fibres pass to the cortex directly, all the lesions of the nucleus lateralis cause degeneration of thalami-cortical fibres.

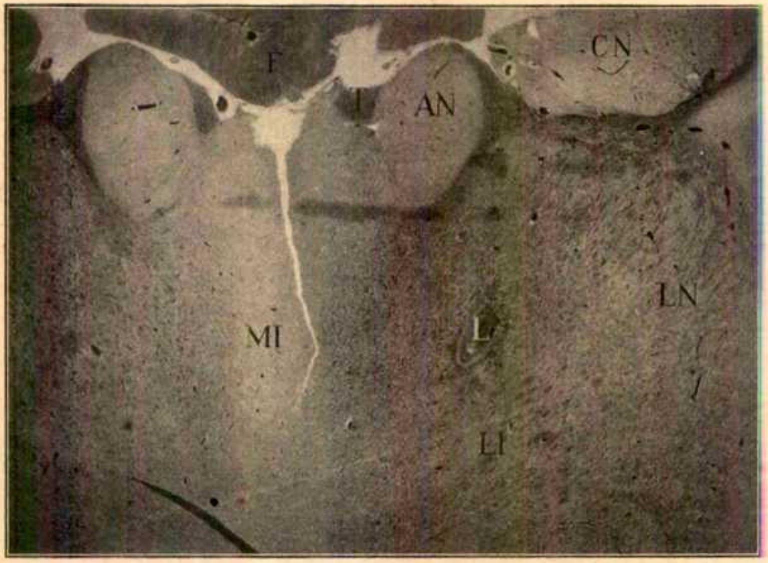


FIG. 33.—[Rh. 289.]

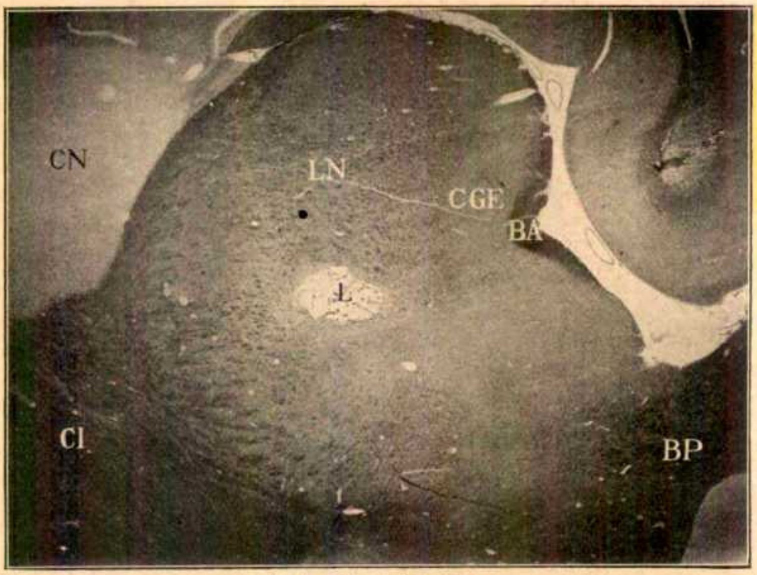


FIG. 34.—[Cat 247.]

The second, that the thalami-cortical fibres are revealed by this method to be arranged dorso-ventrally.

The third is, that the thalami-cortical fibres furnished to the two central gyri differ in number and size very materially.

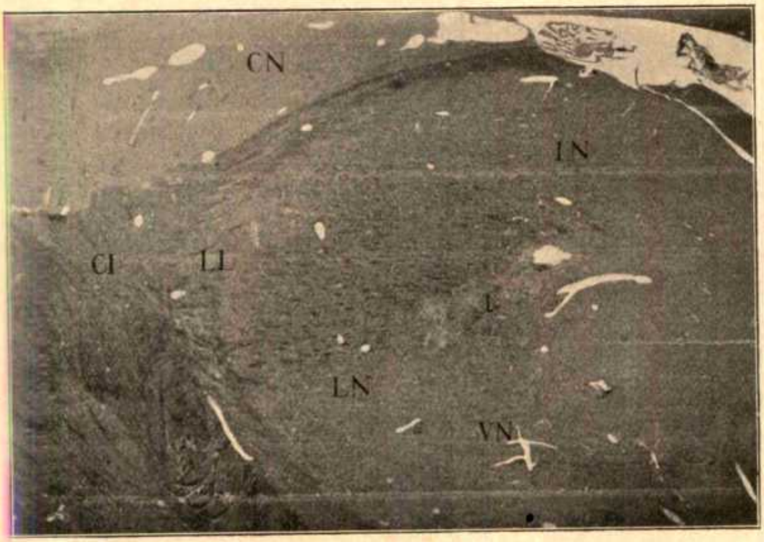


FIG. 35.—[Rh. 232.]

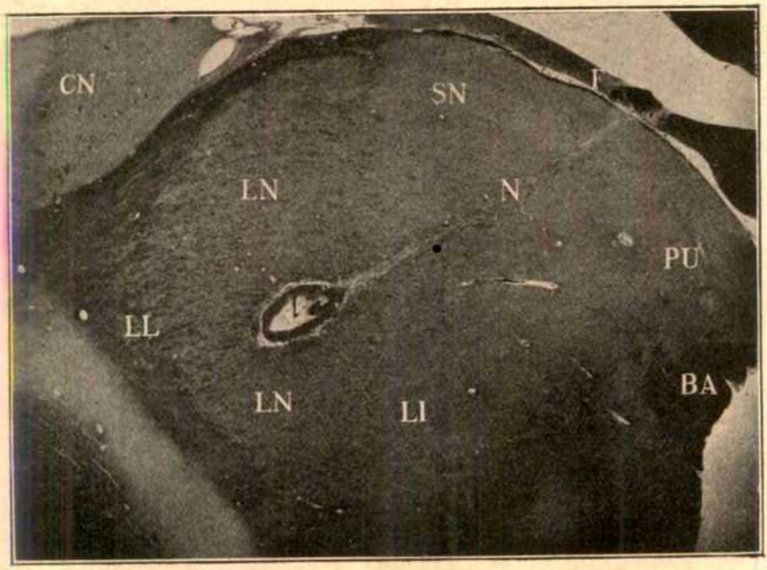


FIG. 36.—[Rh. 270.]

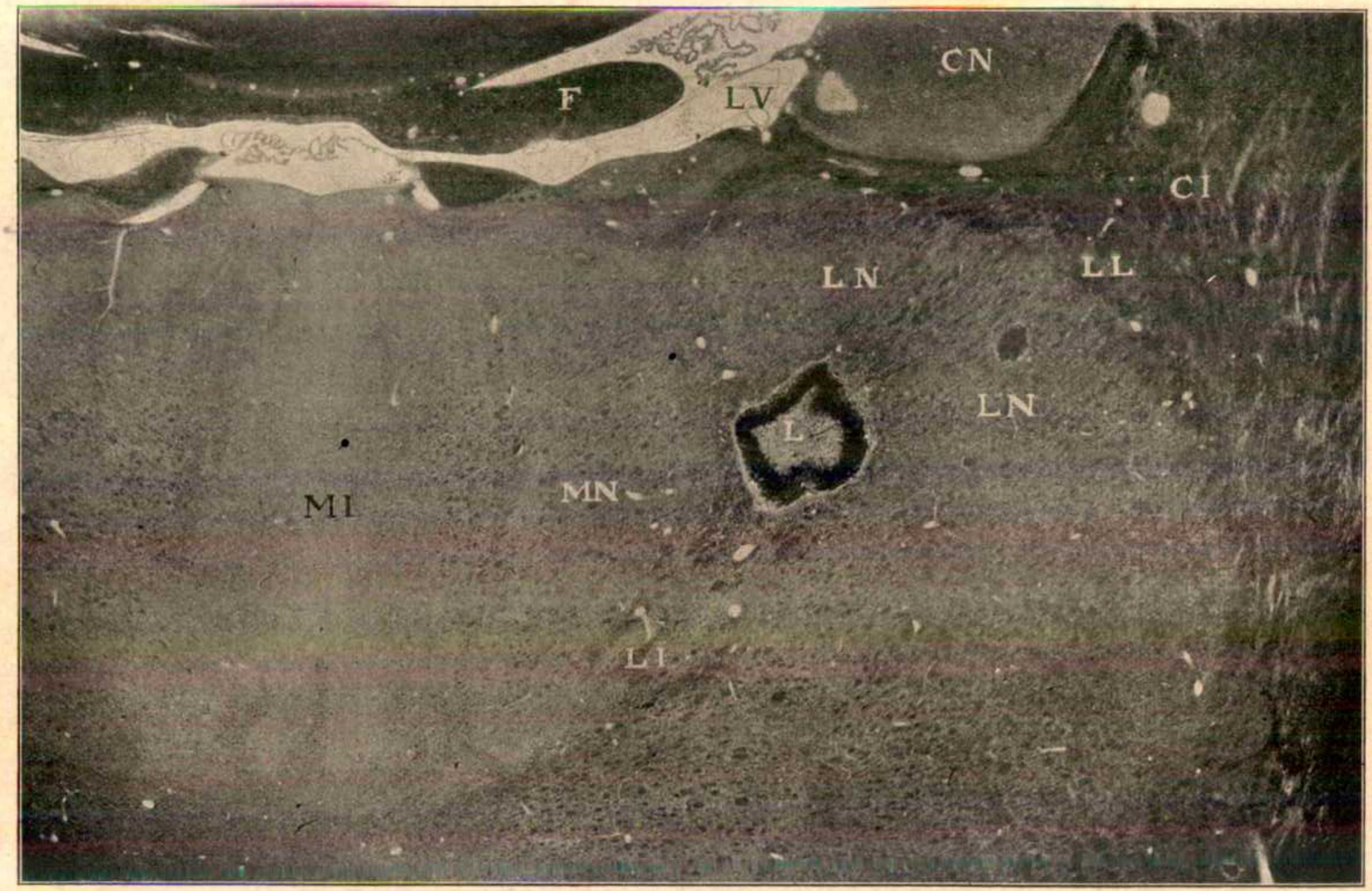


FIG. 37.—[Rh. 240.]

Lesion of inner border of nucleus lateralis, showing clearly both the medial group of inwards-running fibres and the outer group passing to the capsula interna.

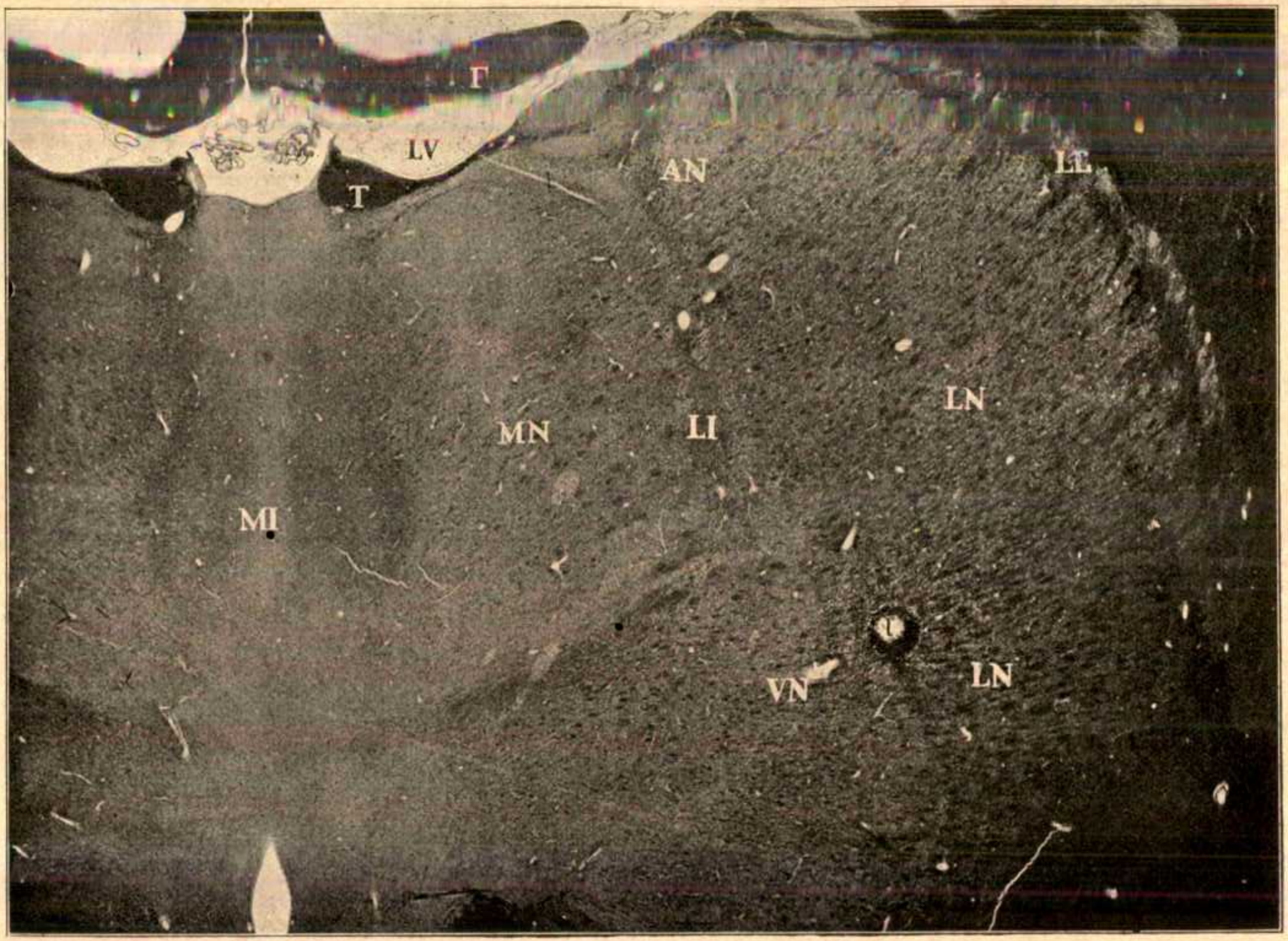


FIG. 38.—[Rh. 272.]

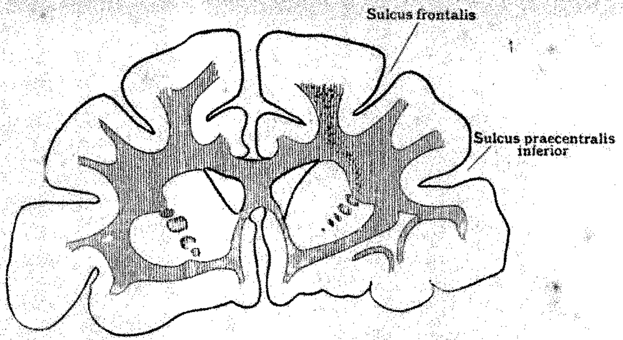


FIG. 39.—Rhesus 261 (I).

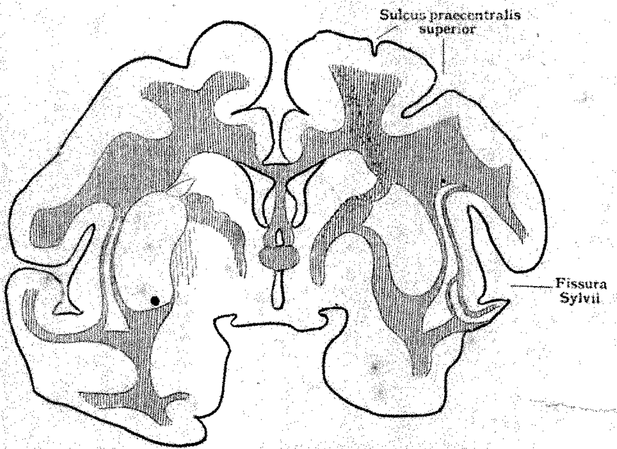


FIG. 40.—Rhesus 261 (II).

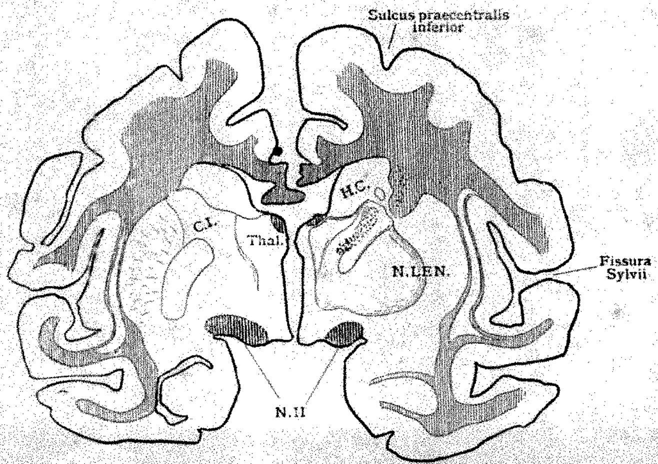


FIG. 41.—Rhesus 261 (III).

The first point confirms the fundamental conception of Burdach before referred to, and will perhaps lead to wider generalizations on the comparative neurology of the thalamic nuclei. From the most anterior portion of the nucleus lateralis fibres proceed, as seen in figs. 39, 40, &c., from Rh. 261, to the gyrus frontalis superior. The

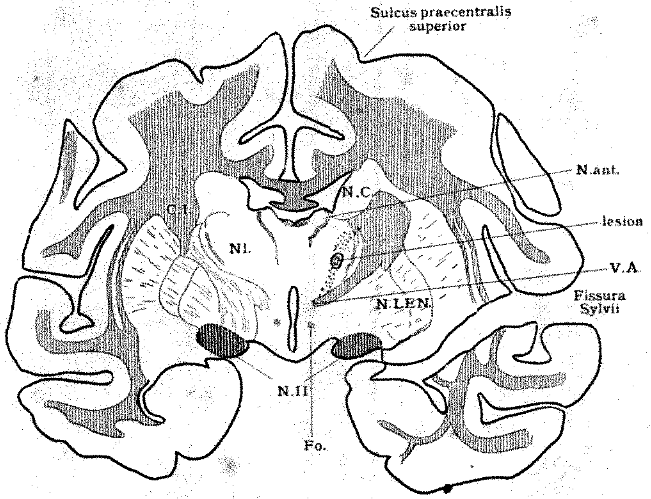


FIG. 42.—Rhesus 261 (IV).

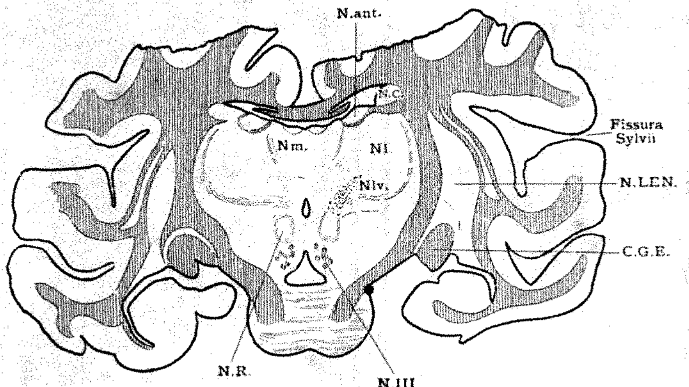


FIG. 43.—Rhesus 261 (V).

second point is one of great practical import, and must receive further attention. In the first place the thalami-cortical fibres derived from the lesions of the ventral region of the lateral nucleus are distributed only[1] to the so-called motor area and the post-central gyrus.

[1] It will be noted that this statement is conditioned by reference to the exact lesions photographed. It is, of course, left open whether the neighbouring parts of the nucleus lateralis send fibres to regions of the cortex other than those specified.

Furthermore, from the dorsal part of the nucleus lateralis thalami-cortical fibres pass in numbers to the lower limb area of the cortex, from the middle part to all three areas—lower limb, upper limb, face; while from the lowest lesion of all the degeneration can be traced into the operculum and facial centre only.

This dorso-ventral topographical order of representation is what might be expected to exist, but the strikingly sharp delimitation of the face from the limb areas as receptors of thalami-cortical fibres could not have been justly surmised.

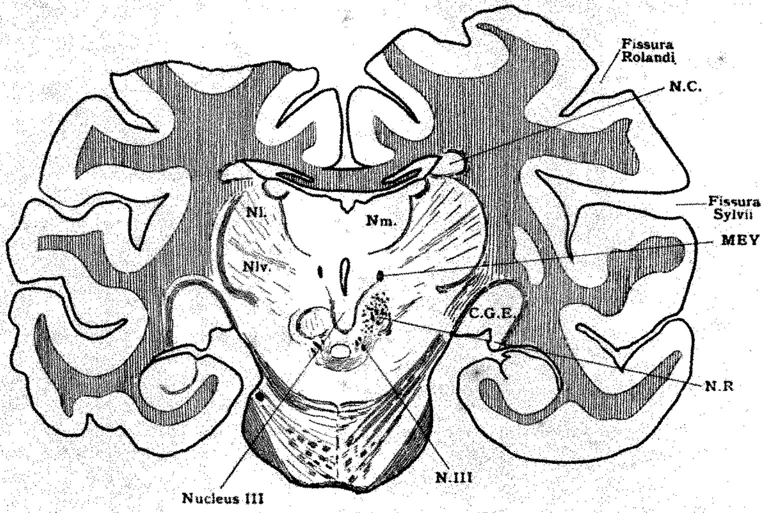


FIG. 44.—Rhesus 261 (VI).

Reference to figs. 45-48 will show better than description how extremely sharply defined is the supply of thalami-cortical fibres to

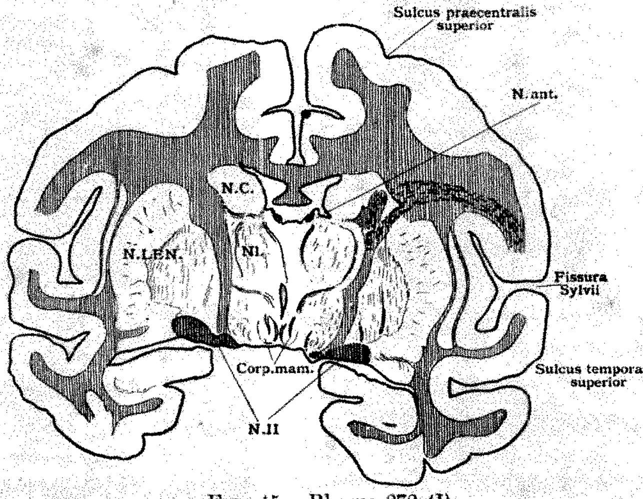


FIG. 45.—Rhesus 272 (I).

the face area, and how this affords a contrast to the associated supply of the two limb areas.

As regards the recent work by Brodmann and Campbell [29] on the distribution of the large Betz cells of the so-called motor region, it will be seen that in part this distinction between the two groups of thalami-cortical fibres follows the grouping of the large pyramidal cells, but more cannot be said on this point as yet. It, however, leads naturally to the next question, the third named above—*i.e.*, the distinction in number and size of the thalami-cortical fibres supplying the pre-central and post-central gyri respectively. From sagittal section-series it was at once

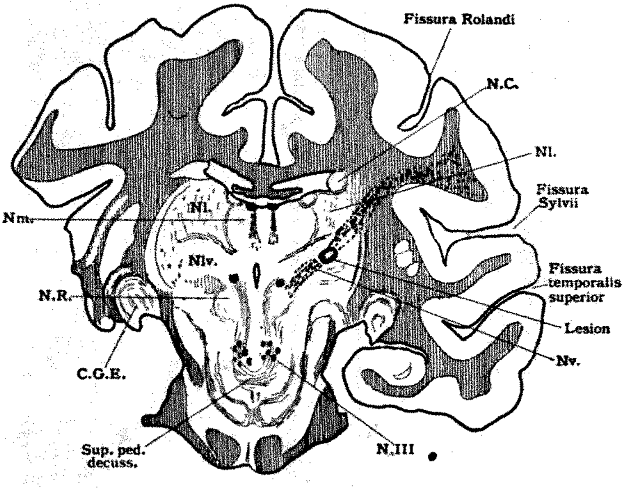


FIG. 46.—Rhesus 272 (II).

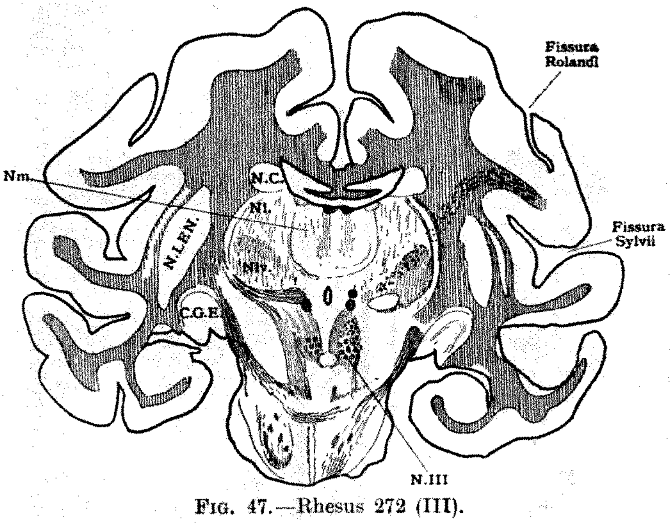


FIG. 47.—Rhesus 272 (III).

evident that far more thalami-cortical fibres proceeded to the pre-central than to the post-central gyrus. This discovery was contrary to preconceived opinions, and therefore was investigated with special care. It then became clear that, besides the question of number of fibres, their relative diameter must be taken into account; and it was found that, whereas the pre-central received a large number of fibres, a few of which were large, very many medium, and some fine, the post-central received fine fibres, with a very few medium ones.

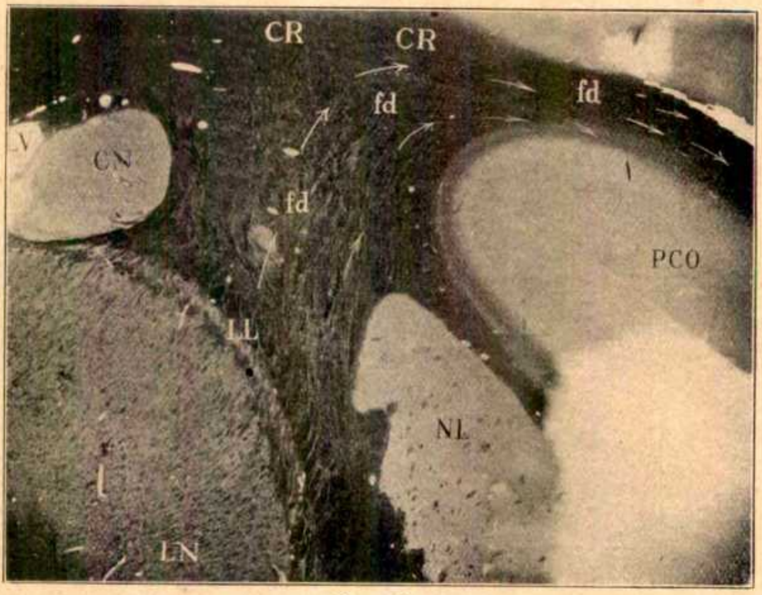


FIG. 48.

Clearly there is a definite morphological difference in the supply of fibres to the two gyri from the optic thalamus, and the net result is that a larger number and larger-sized fibres pass to the pre-central gyrus (gyrus frontalis ascendens).

Group II.—Mesial Fibres.

In three cases[1] it was found that from the inner side of the lesion medium-sized and fine fibres passed to the "centre median" and the nucleus arcuatus (*see* figs. 35-37). This is especially important, as the lesions furnishing these connection paths are all situated in the dorsal third of the nucleus lateralis. A certain degree of fine degenerated fibres and collaterals (?) can be seen extending all around the lesions for about one-fourth of the diameter of the nucleus.

---

[1] Rh. 228, 232, 240.

Lamina Interna.

In two cases—Rh. 111 and Rh. 289—the lesion was situated in the lamina interna, and caused consequently a limited destruction of the inner margin of the lateral nucleus.

IV.—LESIONS OF LAMINA INTERNA AND ADJACENT NUCLEUS LATERALIS.

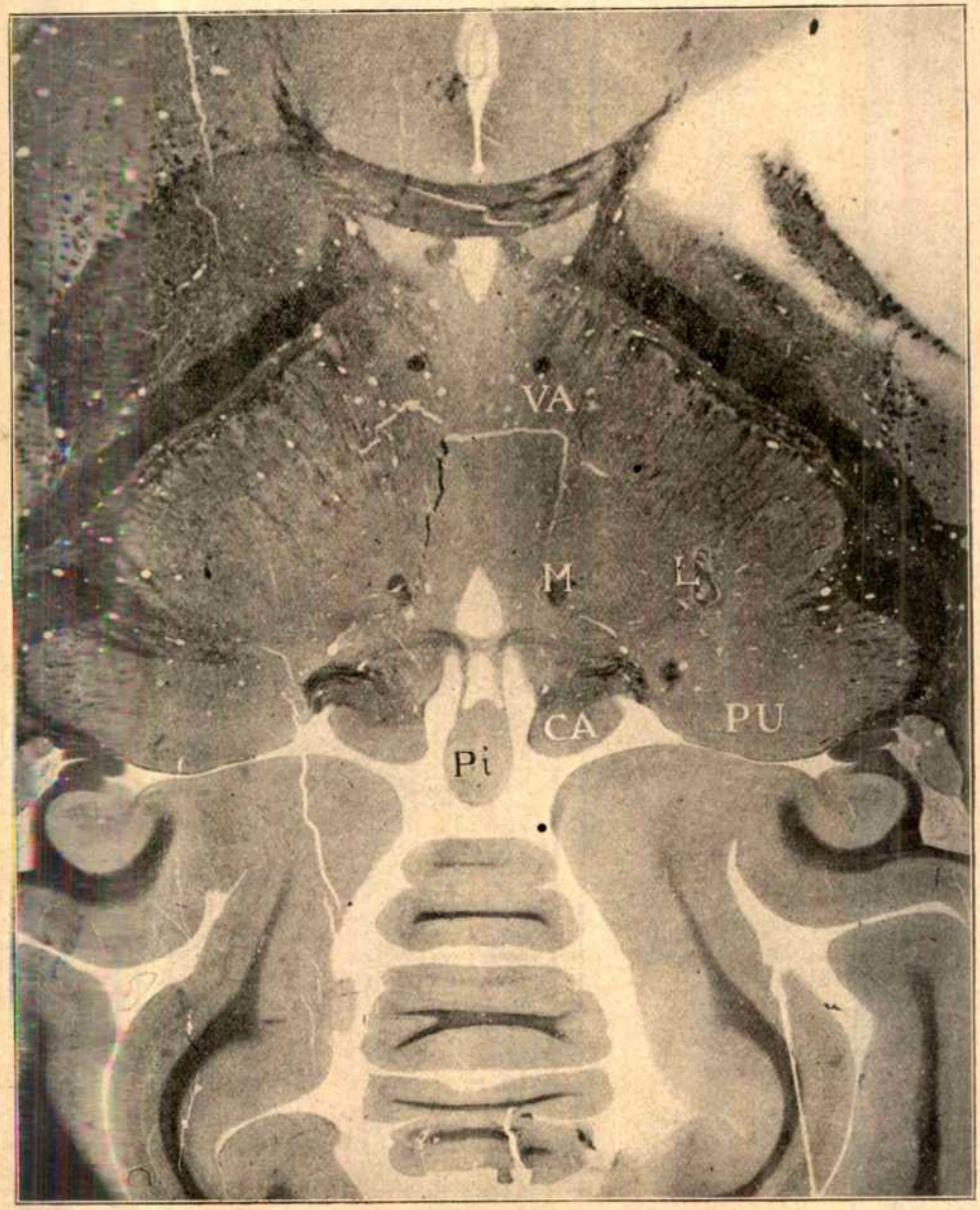


FIG. 49.—[Rh. 267.]

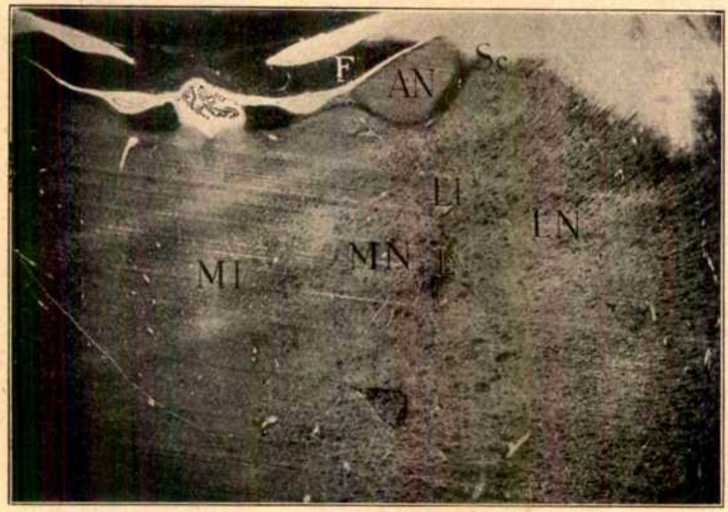


FIG. 50.—[Rh. 111.]

The resultant degenerations were the same as those of a primary and restricted lesion of the lateral nucleus.

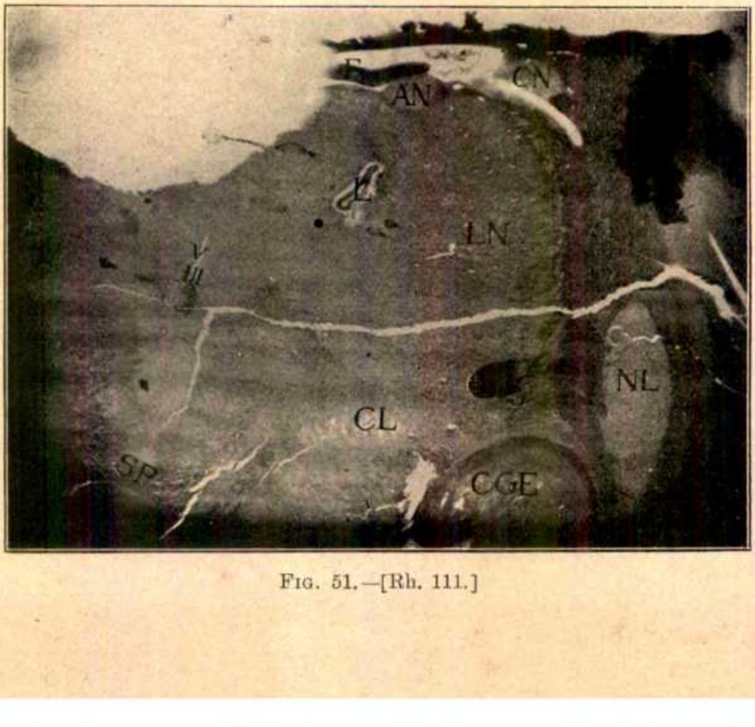


FIG. 51.—[Rh. 111.]

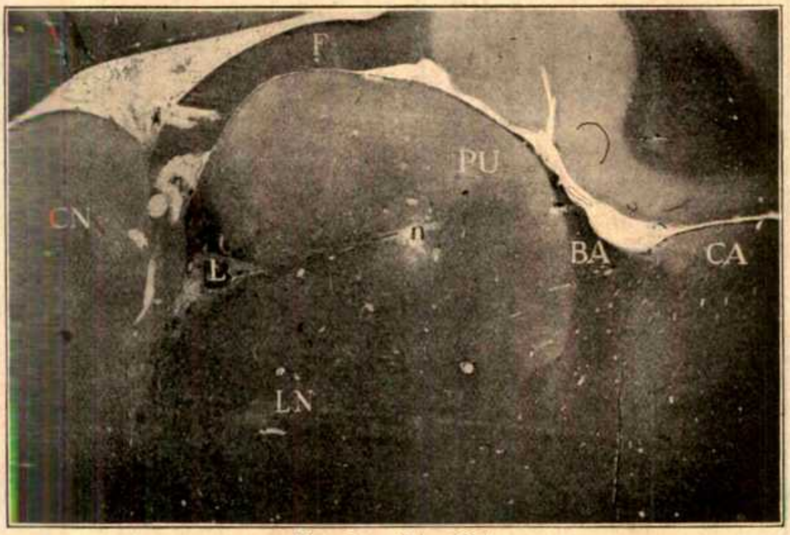


FIG. 52.—[Rh. 255.]

Lesion of upper angle of nucleus lateralis dividing also the tænia thalami.

V.—LESIONS OF CAPSULA INTERNA AND ANTERIOR MARGIN OF NUCLEUS LATERALIS.

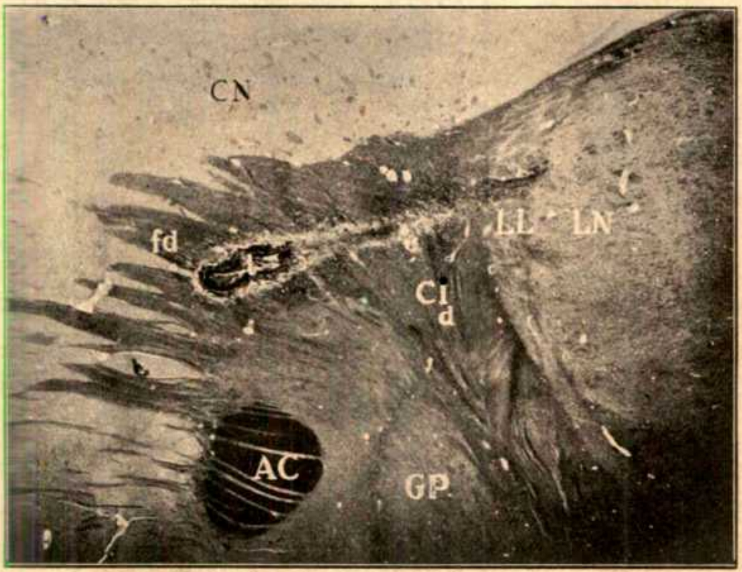


FIG. 53.—[Rh. 258.]

Lesion of genu and pars anterior of capsula interna.

fd = thalami-subcaudate fibres degenerated.
d = pyramidal (descending) fibres degenerated.

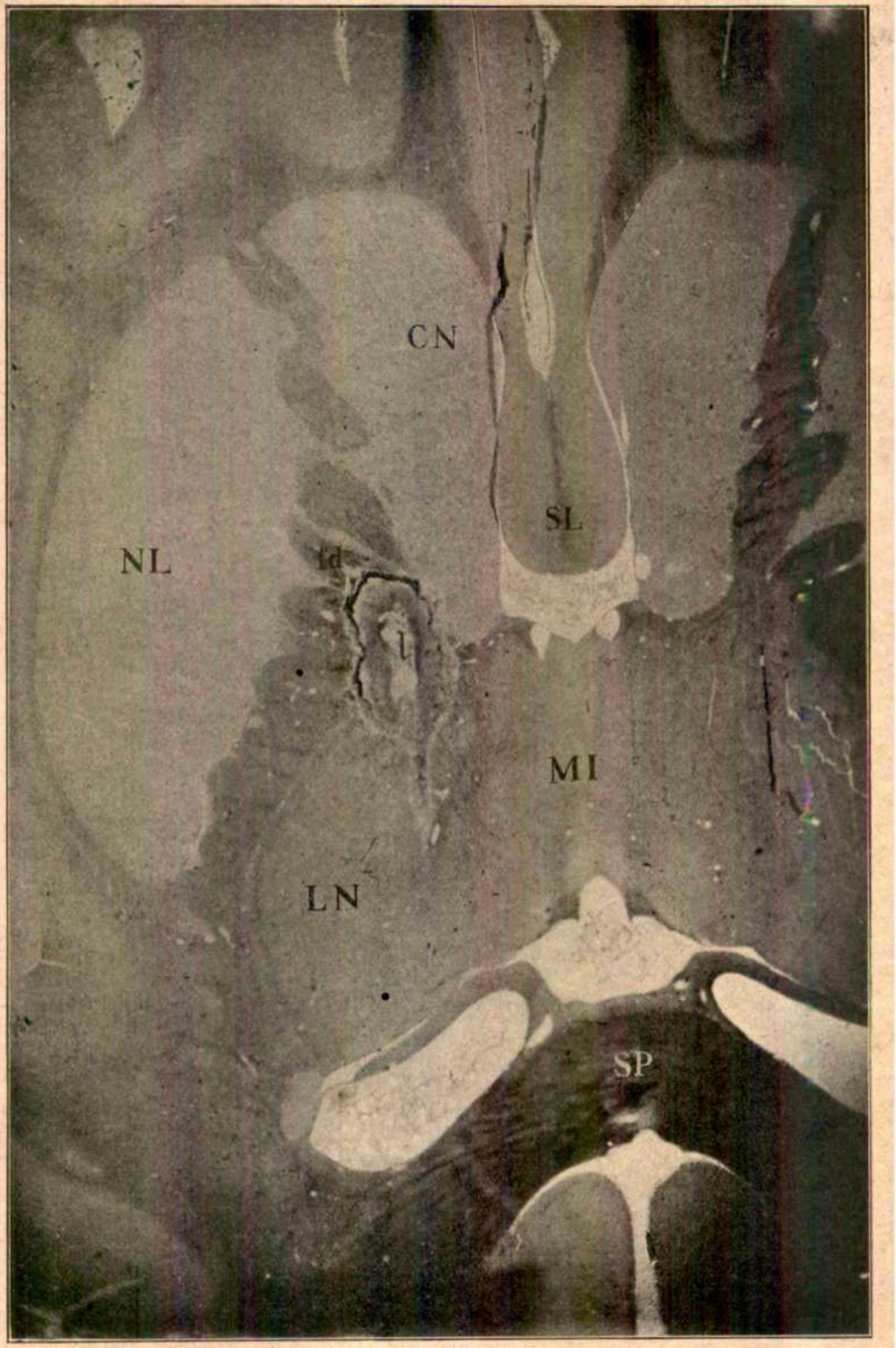


FIG. 54.—[Rh. 236.]

Note the considerable mass of thalami-cortical fibre degeneration.

Centre Median and Nucleus Arcuatus.

These two nuclei have been grouped together as they are closely connected with one another, and seem to receive similar fibre supplies. This idea has been prevalent for some years, though no positive proof in support thereof has hitherto been furnished.

Lesions in this region were successfully made in five animals. In Rh. 216 and Rh. 218 the lesions were confined almost exclusively to the nucleus arcuatus; in Rh. 275 the centre median alone was injured; in 257 the border between the centre median and nucleus lateralis was injured, but so slightly that none of the degenerations characteristic of a lateral nucleus lesion—as described in the preceding pages—were observed. In Rh. 87 both the centre median and nucleus arcuatus were destroyed, as well as a considerable portion of the nucleus ventralis.

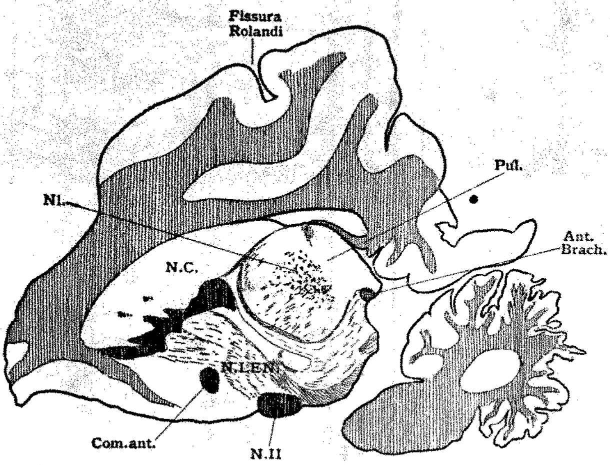


FIG. 55.—Rhesus 275 (I).

Three principal facts are observed in these cases:—

First.—The complete absence of thalami-cortical fibres. Not only were no fibres ever seen going to the cortex, but all fibres from centre median and arcuate lesions stopped in the inner half of the lateral nucleus—*i.e.*, before they reached the internal capsule (*see* figs. 55, 56).

Second.—That all the fibres go to the nuclei immediately adjacent, and that these fibres all run a short course, yet are of medium calibre. When the nucleus arcuatus was involved, as in Rh. 216, 218, and 87, fibres passed from it to all the thalamic nuclei, with the exception of the

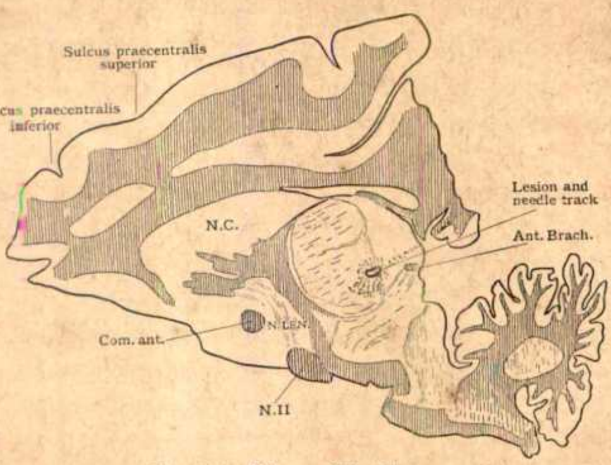


FIG. 56.—Rhesus 275 (II).

VI.—LESIONS OF CENTRE MEDIAN AND IMMEDIATELY ADJACENT REGIONS.

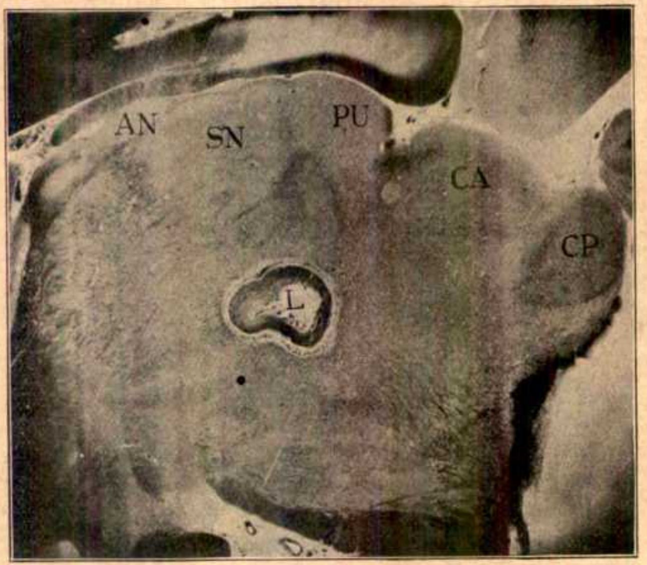


FIG. 57.

nucleus anterior. When the centre median was alone involved, as in Rh. 275 and Rh. 267, no fibres entered either the median or anterior nuclei, but many passed to all the other thalamic nuclei.

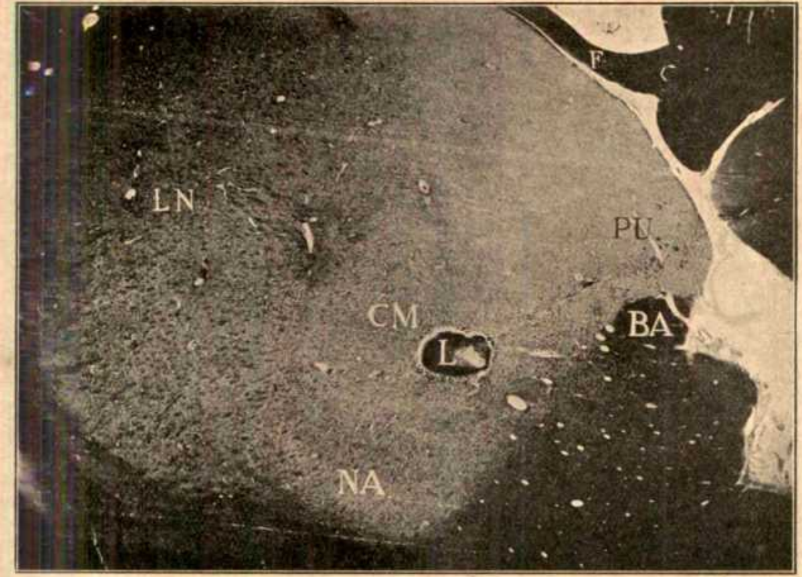


FIG. 58.—[Rh. 273.]

Third.—There were no descending fibres to the mesencephalon.

These facts clearly show that the centre median and nucleus arcuatus are association centres and connect the various nuclei with one

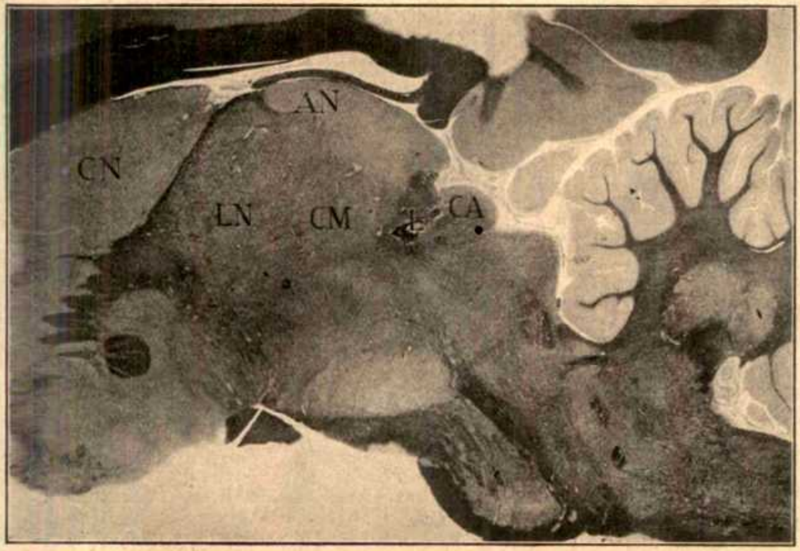


FIG. 59.—[Rh. 273.]

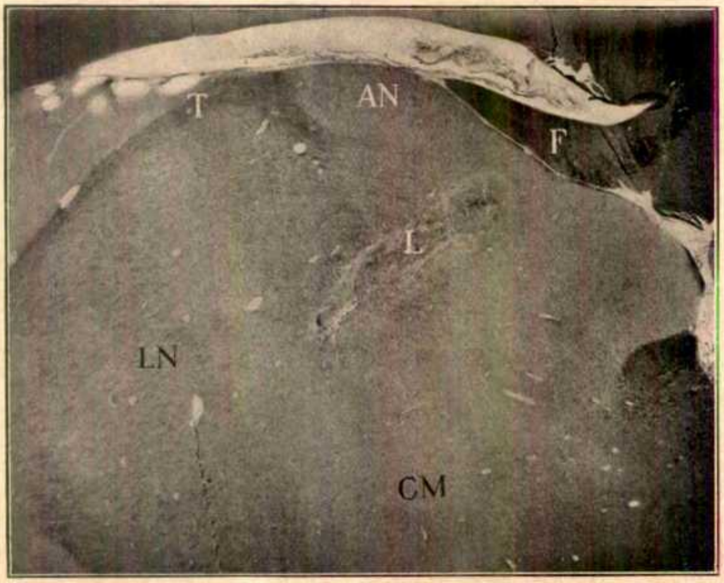


Fig. 60.

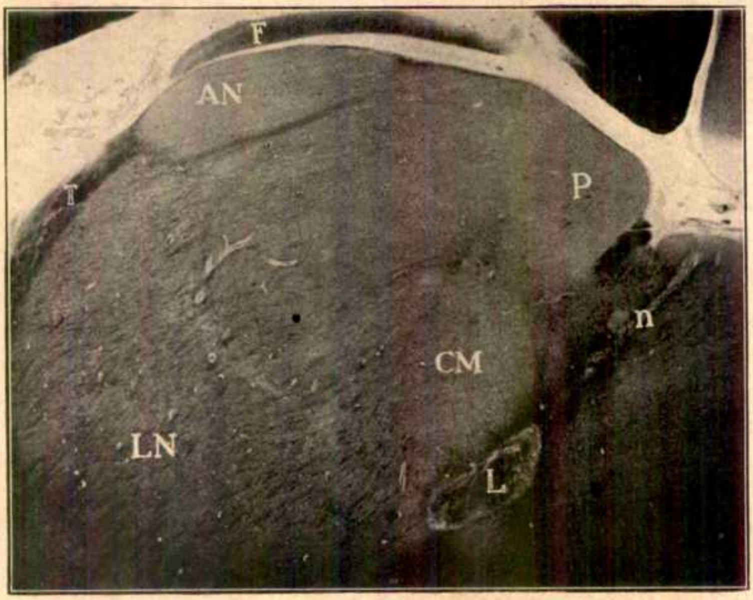


Fig. 61.

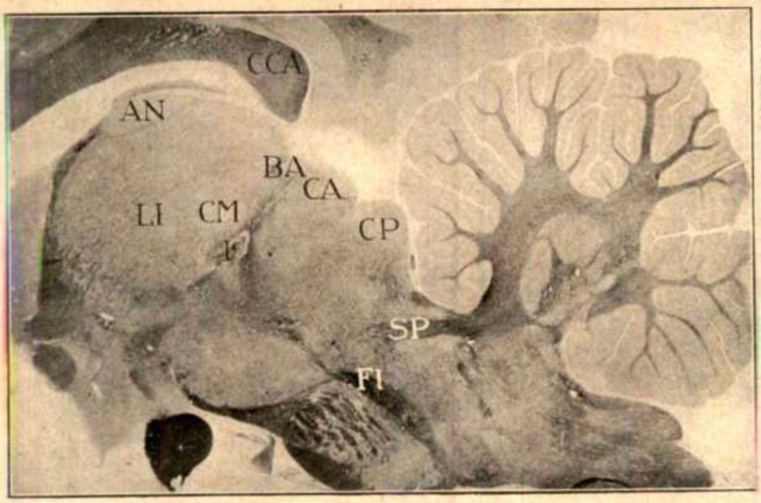


FIG. 62.—[Rb. 218.]

another, though they seem to be much more intimately associated with the lateral part of the thalamus than with the median portion. The advisability, therefore, of keeping the original name suggested by Luys seems apparent.

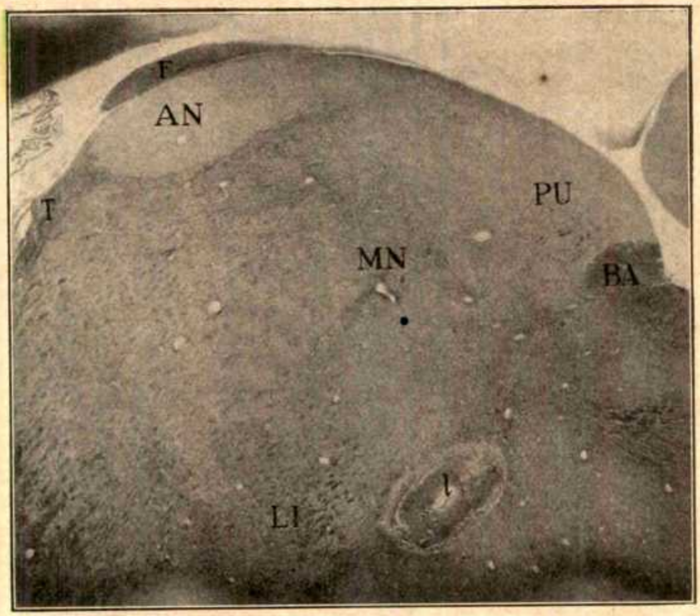


FIG. 63.

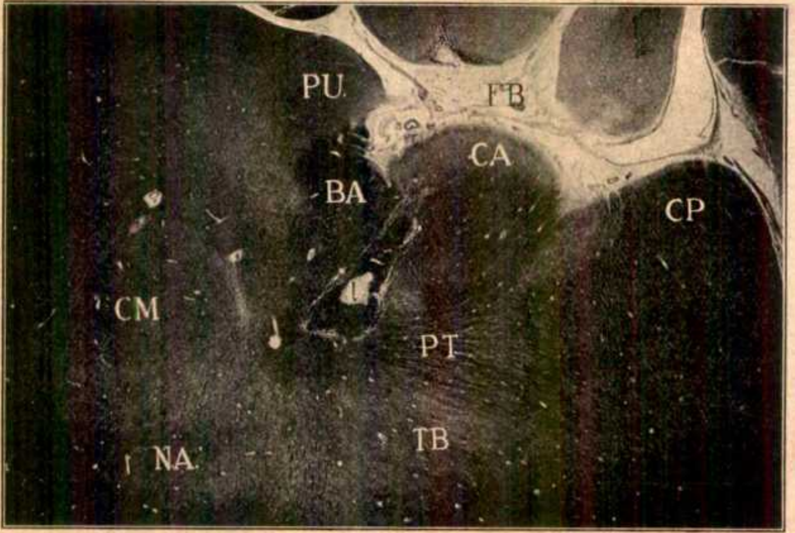


FIG. 64.—[Rh. 273.]

In Rh. 87 a further important point developed, which is well shown on fig. 66. The lesion, as described in Table II, injured the nucleus ventralis. From this region the posterior longitudinal bundle is seen to degenerate downwards, and sends collateral fibres to the following cranial nerves: V, VI, VIII, X.

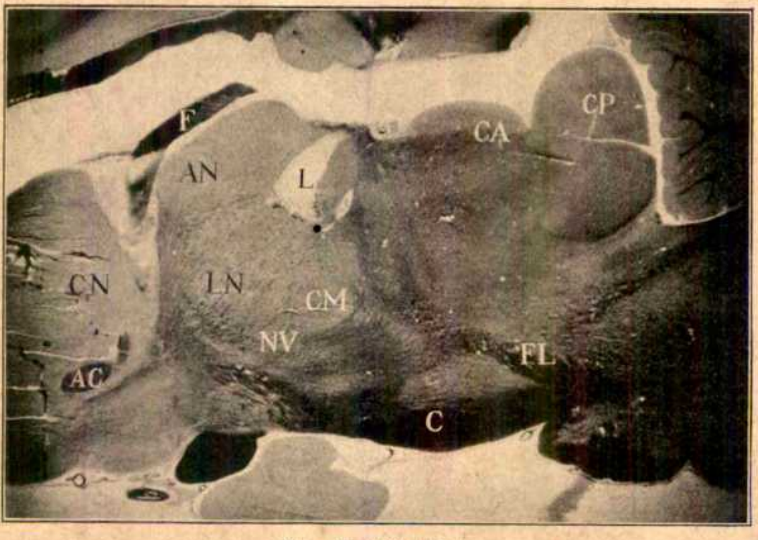


FIG. 65.—[Cat 95.]

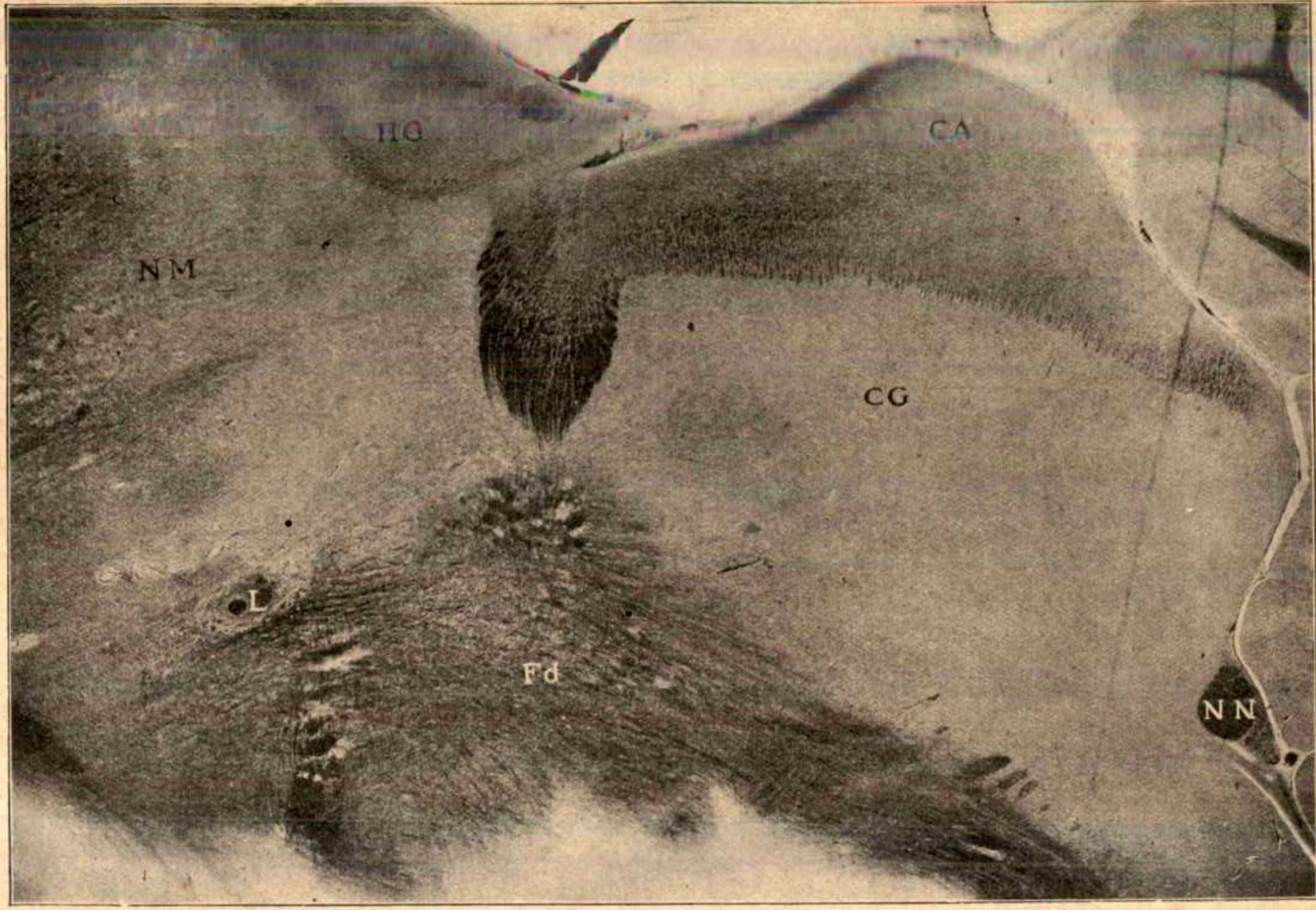


Fig. 66.- [Rh. 87.]

Held [70] and van Gehuchten [62] regarded the posterior longitudinal bundle as a descending tract, while Kölliker [83] and Cajal [28] believed it was an ascending one. The first two observers differed as to the origin of the tract. Held thought it came from the anterior colliculus, while van Gehuchten found it arose from the grey substance just above the third nucleus, which he calls the superior nucleus of the posterior longitudinal bundle. As a matter of fact the bundle contains both ascending and descending fibres, and in the present case evidently the latter portion arises higher than those observers thought—namely, in the ventral nucleus of the thalamus. This is the only instance found in these experiments of any fibres from the thalamic region going into the spinal cord.

The injury in Rh. 87 to Meynert's bundle caused its degeneration down to the interpeduncular ganglion.

Pulvinar.

No lesions were made in the pulvinar alone, since to obtain control observations on this region the needle should be introduced from the front, and, as the technique of this procedure would have involved special alterations of the apparatus, this part of the examination of the thalamus was left to a future research.

Hypothalamus.

In the course of this work several lesions were made in regions adjacent to the thalamus, and as the resultant degenerations throw more light on the relations of the thalamus to the surrounding centres they will be now described.

These cases were Rh. 203, 207, 251, and 211. The first three have many points in common.

In all these the median fillet was injured, either as it passed around the outer side of the red nucleus, or somewhat more dorsally, or again anteriorly, where it begins to spread out into the ventral portion of the lateral nucleus. As a result of these lesions the final distribution of the fillet is well seen, and again proves beyond all question that the view of Tschermak [151], that the fillet fibres end in the cortex, is not correct.

In Rh. 207, where there was a slight injury to the ventral nucleus, a few fibres could be traced towards the opercular region, where they branch off from the internal capsule above the island of Reil. As they were not traced to the cortex the observation is incomplete, but they tend to confirm the findings in Rh. 272. Except for the fillet fibres, no other fibres pass to the thalamus from Forel's field, which was involved in each of these lesions, and in Rh. 207 the few fibres entering the arcuate and centre median arise from the point of injury of the ventral nucleus.

The lesions may now be considered in detail under the head of (*a*) Forel's field; (*b*) Nucleus ruber.

(*a*) *Forel's Field.*

Of the three cases of lesions in this region, Rh. 203 and 207 exhibited the focus as situated in the junction of the outer and middle thirds of the more dorsal part of the field, while in Rh. 251 the lesion

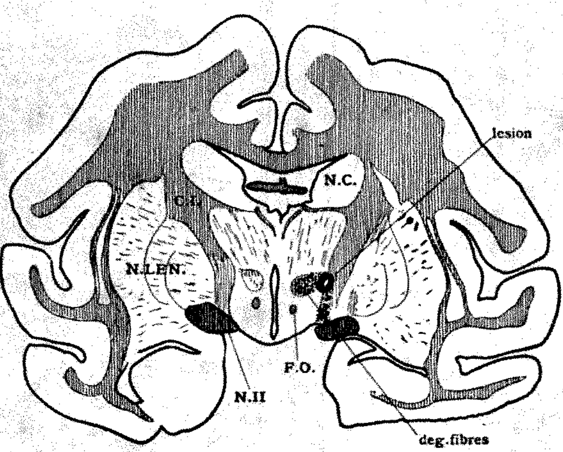


FIG. 67.—Rhesus 251 (I).

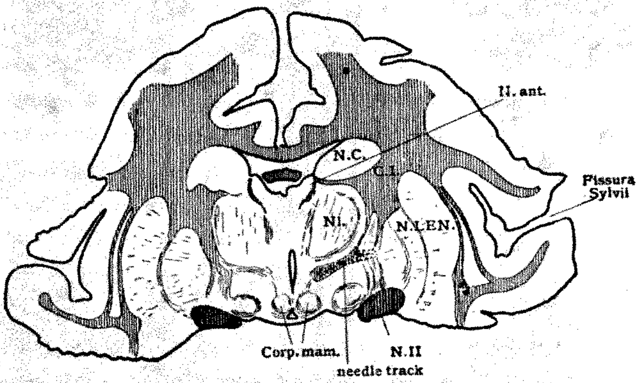


FIG. 68.—Rhesus 251 (II).

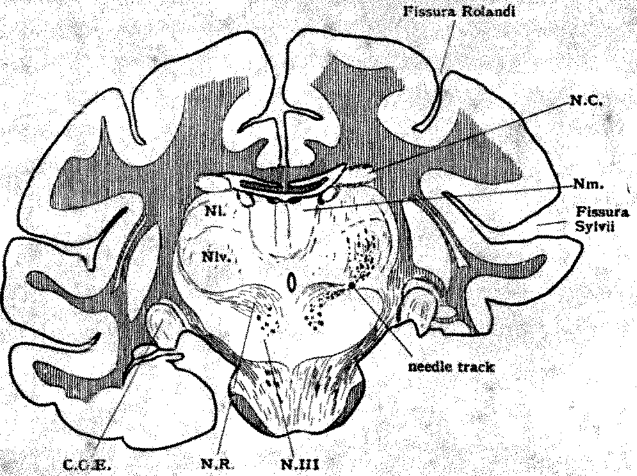


FIG. 69.—Rhesus 251 (III).

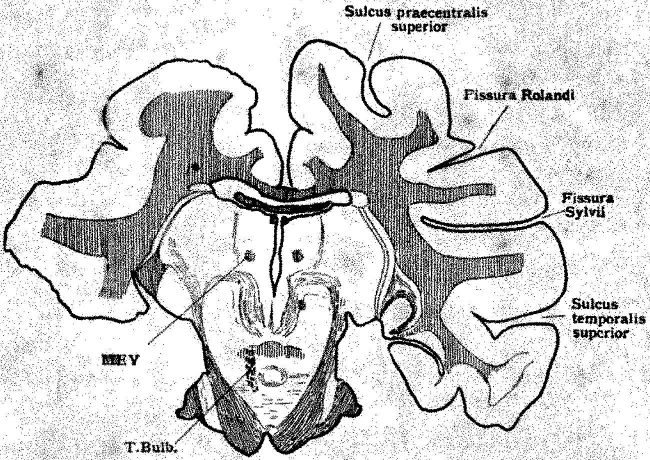


FIG. 70.—Rhesus 251 (IV).

was situated in the middle of the field, slightly ventrally of an equatorial line. In this case anteriorly the lesion involved the base of the internal capsule. Apart from the concomitant consequences above described of injury to the neighbouring fillet or ventral nucleus, there were to be seen two groups of degenerated fibres proceeding horizontally from the lesion and forming a lateral and a mesial group (*see* figs. 67-70).

Group I.—Lateral Fibres.

These were coarse and medium-sized fibres which passed horizontally outwards in a well-marked bundle interlacing at right angles with the basal third of the internal capsule, and entering the globus pallidus, in which they broke up. A few reached the middle zone of the nucleus lenticularis, but none passed into the putamen.

Group II.—Mesial Fibres.

The mesial degeneration consisted of (1) a very interesting mass of fine and medium-sized nerve fibres forming the "inferior peduncle of the thalamus" (Dejerine) (*see* figs. 67, and 71 and 72 FOR), but which is in essence the frontal section of the ansa lenticularis; (2) coarse and medium fibres to the nucleus ruber, &c.

(1) Of these the fine fibres in the degenerated ansa group pass backwards (none forwards); some give fine collaterals to the side of the tuber cinereum (none internal to the fornix), but the main body continue spreading over the whole of the medial part of the field of Forel and sending numerous fine collaterals or ending in the ventral nucleus. Finally, the remainder cease in the anterior capsule of the red nucleus.

(2) The fibres which pass down to the nucleus ruber break up in its inferior capsule, and in a few instances cross the commissure of Forel and gain the capsule of the opposite nucleus ruber.

(*b*) *Nucleus Ruber.*

In one experiment [Rh. 211], the lesion was placed in the ventral half of the dorsal (anterior) nucleus of the nucleus ruber (*see* fig. 75).

The fibres observed to degenerate therefrom are divisible into a lateral and a mesial or ventral group, as in the case of the other nuclei.

(1) *Lateral Fibres.*—A small number of medium-sized fibres passed outwards and upwards into the lateral nucleus and into the centre median. According to Horsley and Clarke these are fibres of the superior cerebellar peduncle, *i.e.*, fibres in transit, but whether any also arise within the injured nucleus cannot be decided.

(2) *Mesial Fibres.*—These are traceable downwards and inwards, crossing in Forel's commissure and divisible into two groups, as described in Table II, viz., rubro-spinal and rubro-bulbar. The former, being slightly marked by but a few fibres, require no discussion, but it is important to point out that the rubro-bulbar tract observed in this case is naturally not the same as that described by Münzer and Wiener [112] as tractus-tecto-bulbaris, but lies rather further from the median plane.

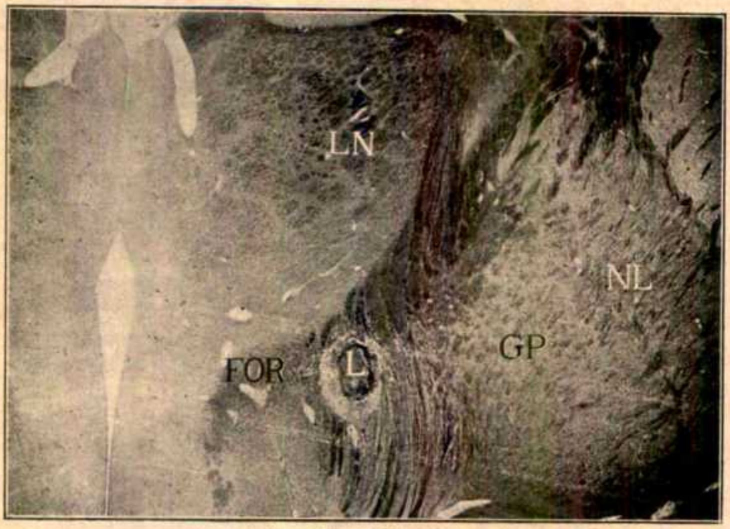


FIG. 71.—[Rh. 251.]

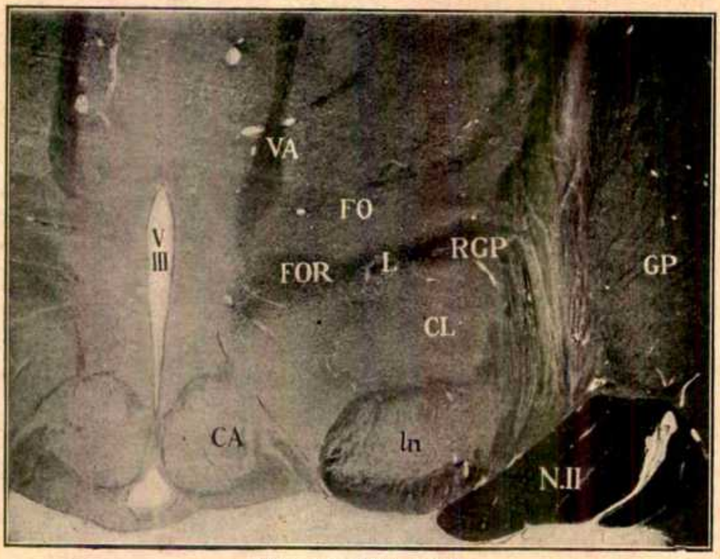


FIG. 72.—[Rh. 251.]

Section further caudal to preceding, showing the globus pallidus fibres RGP crossing the lower part of the genu of the capsula interna.

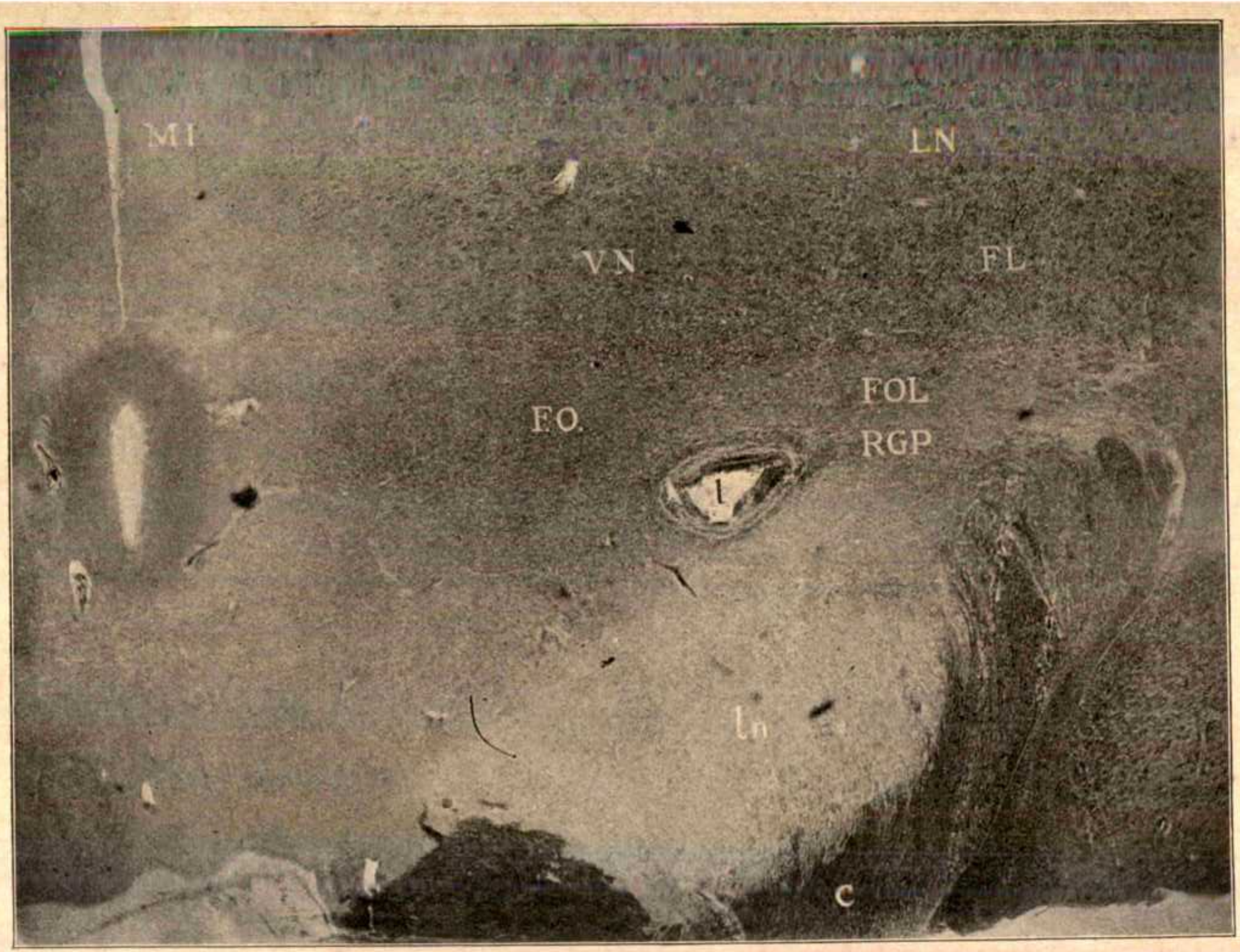


FIG. 73.—[Rh. 13.]

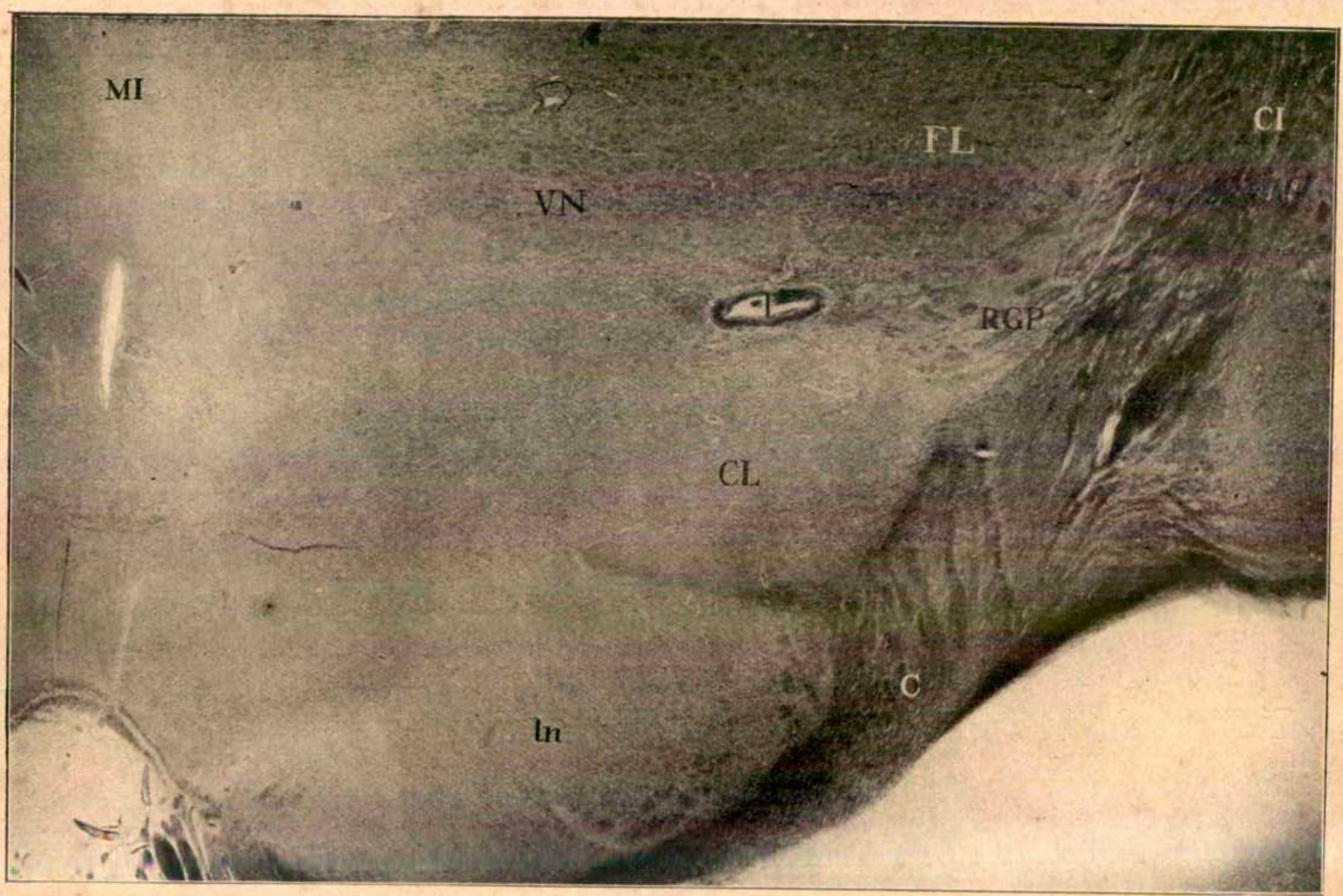


FIG. 74.—[Rh. 207.]

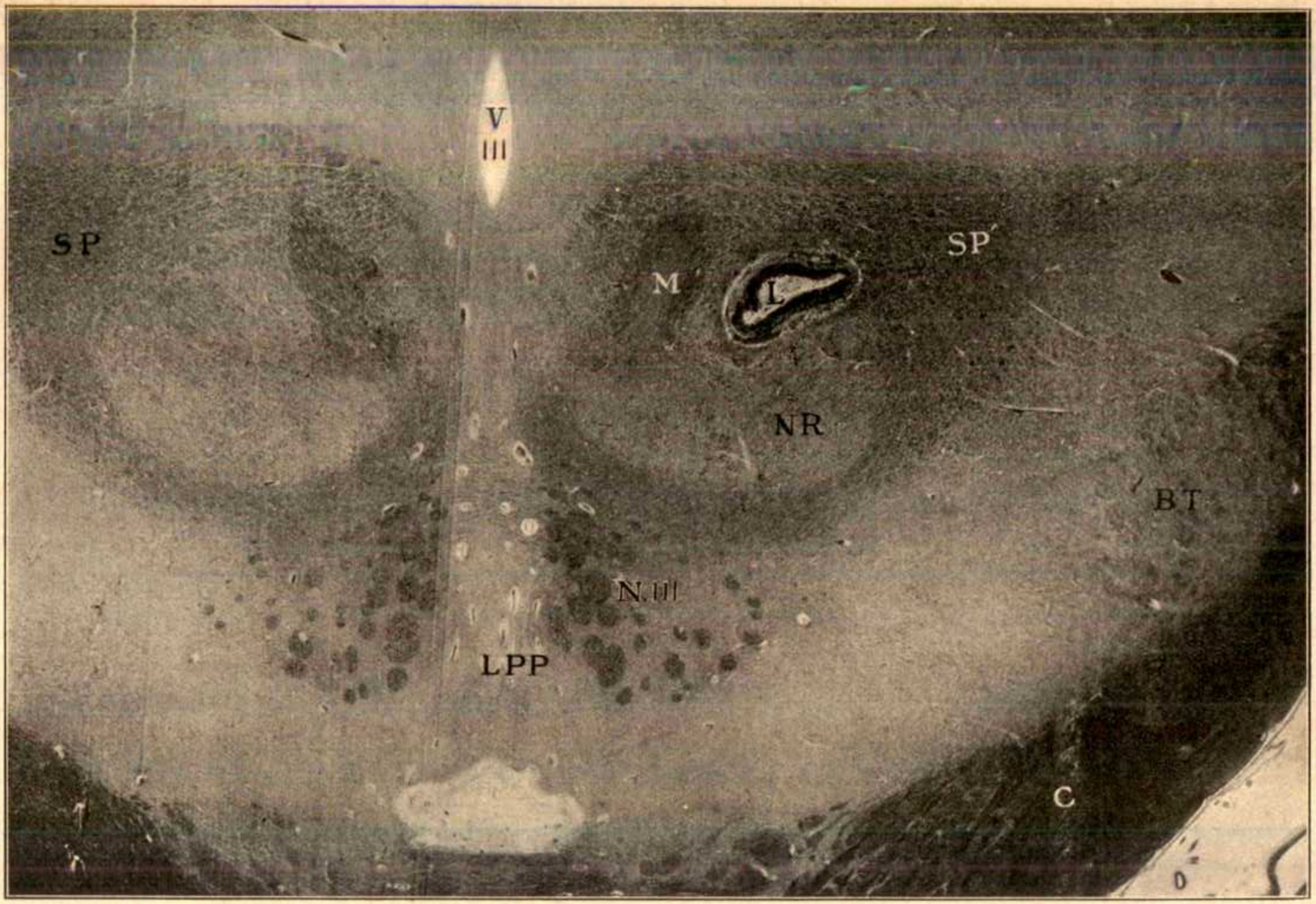


FIG. 75.—[Rh. 211.]

TABLE II.—*Thalamic Lesion.* (*Rh.* = *Macacus rhesu*

Lesions

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus ve tralis
Rh. 189	Nucleus anterior	Fine fibres degenerated in anterior limb of internal capsule close to caudate ending indeterminate	Lesion	Fine fibres break up here in outer border running back from lesion	Nil	Nil	Nil ...
Rh. 222	Nucleus anterior and Vicq d'Azyr's bundle just below the nucleus	Nil	Lesion; a few fibres from injury to Vicq d'Azyr's bundle seen entering nucleus anterior	Indeterminate	Nil	Nil	Nil ...
Cat 255	In anterior part of fornix; Vicq d'Azyr's bundle just before it enters nucleus anterior, needle track passes through nucleus lateralis	Fibres to cingulum along its entire length; a few coarse fibres to gyrus lateralis, suprasylvian and anterior ectosylvian from needle track, finer fibres through the caudate chiefly at its anterior and outer pole, to gyrus cruciatus posterior	Fine fibres break up here; they come from Vicq d'Azyr's bundle	Nil	Lesion ...	Nil	Nil ...
							Lesions
Rh. 111	Involves outer border of median nucleus, lamina interna, and internal portion of lateral nucleus	Leg and arm area of precentral gyrus and about to junction of middle and lower thirds of precentral gyrus, part of face centre, most fibres to leg centre	Needle track passes through capsule of nucleus; a few fibres from lesion posteriorly ascend to lower border of anterior nucleus and appear to end there	Lesion; fine fibres break up in posterior part of nucleus	Lesion ...	Nil	Nil ..

ummary of Degenerations Observed.

ucleus Anterior

ntre median nd nucleus arcuatus	Pulvinar	Nucleus ruber	Descending fibres to medulla and pons	Cord	Fibres elsewhere.	Remarks.
1	Nil ...	Nil	Nil	Pyramidal degeneration due to needle track	To caudate ...	Needle introduced vertically.
1	Nil	Nil	Nil	Nil	All end in caudate	This lesion was due to an embolus which lodged in the nucleus anterior and caused a small necrosis. Through a miscalculation the electrolysis occurred in the ventricle in front of anterior colliculus, and from here the vessel in which embolus occurred can be traced. Needle introduced horizontally.
1	Nil	Nil	Nil	Nil	Nil	Needle introduced horizontally.

ucleus Medius

... ...	Nil... ...	Nil	Nil	Pyramidal fibres degenerated due to passage of needle	Some fibres from nucleus lateralis go to outer part of ganglion habenulæ	Needle introduced horizontally.

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus ve-tralis
Rh 169	Median nucleus ..	Nil	Nil	Lesion in outer half, but separated from lamina interna by a considerable portion of uninjured nucleus	Fibres pass through dorsal part just below nucleus anterior	Nil	Nil ...
Cat 95	Median nucleus ventral third of pulvinar posterior limit is Meynert's bundle, which is involved	Pre- and post-crucial gyri and olfactory tubercle	Nil	Lesion ...	Internal surface just touched	Nil	Nil .
Rh. 288	Median nucleus in its upper and outer portion, quite free of the lamina interna	Nil	Nil	Lesion ...	The fibres coming out of the nucleus medius pass obliquely across the superior portion (dorsal fourth); some may end here	Nil	Nil ..

Lesions of Nucl

Rh. 228	Ventral border of dorsal third of nucleus lateralis and most anterior portion of pulvinar	Pre- and post-central gyri arm and leg area; fewer fibres to face region. More fibres to gyrus precentralis than postcentralis	Nil	Medium fine degeneration; source either nucleus lateralis or lamina interna	A few fibres to nucleus of opposite side	Nil	Nil ...
Rh. 232	Between dorsal and middle third of nucleus lateralis	Large number of fibres to precentral gyrus leg and arm area; extremely few and finer calibre fibres to face area and postcentral gyrus	Nil	Nil	Lesion ...	Nil	Nil ..
Rh. 240	Junction of dorsal and middle third of nucleus lateralis	Many fibres to leg and arm area of precentral gyrus; some fibres to postcentral gyrus; fine fibres to superior and middle frontal gyri as far forward as the plane of anterior end of caudate nucleus	Nil	Nil	Lesion; fine fibres seen all through nucleus in neighbourhood of lesion	Nil	Nil ...

ntre median nd nucleus arcuatus	Pulvinar	Nucleus ruber	Descending fibres to medulla and pons	Cord	Fibres elsewhere	Remarks
res cross mina in-rna and reak up ere	Nil	Nil	Nil	Pyramidal degeneration due to passage of needle	Fibres running across dorsal part of lateral nucleus end in caudate nucleus; fibres degenerated due to injury of tænia thalami run back and end in outer half of ganglion habenulæ	As needle introduced vertically cortical fibres obscure the picture and make it impossible to express an opinion as to thalami-cortical fibres, though fibres from median all seem to have ended in caudate.
... ...	Partial lesion	Nil	Nil	Nil	Meynert's bundle degenerated to interpeduncular ganglion	Needle introduced vertically.
l	Nil ...	Nil	Nil	Nil	Fibres enter capsule on lower border of caudate and break up there	Needle introduced vertically.

teralis (A) *Dorsal Third.*

edium-zed fibres reak up erein	Nil	Nil	Nil	Nil	To external geniculate of opposite side (needle track passed through splenium)	Needle introduced horizontally.
me fine bres break p herein	Nil ..	Nil	Nil	Nil	Nil	Needle introduced horizontally.
me fine bres break p herein	Nil .	Nil . ..	Nil	Nil	Nil	Needle introduced horizontally.

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus ve tralis
Rh. 289	Needle track and posterior part of lesion in lamina interna; anterior part of lesion in dorsal and internal third of nucleus lateralis. It occupies an area one fourth the cross diameter of lateral nucleus	Fibres to dorsal half of precentral gyrus as far forward as the superior precentral sulcus and to the angle in front of the inferior precentral, *i.e.*, around the posterior end of the inferior frontal sulcus	Nil	A few fibres run backwards and downwards and break up in outer part of median nucleus	Lesion as described	Nil	Nil ...

Lesions of Nucle

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus ve tralis
Rh. 261	Middle third and anterior part of nucleus lateralis	Fibres to precentral gyrus; arm and leg area; none to operculum, very fine fibres in superior frontal gyrus to plane of anterior end of caudate.	Nil	Nil	Lesion ...	Nil ..	Nil
Rh. 270	Ventral border of middle third of nucleus lateralis	Fibres to pre- and post-central gyri, many fibres to leg and arm area, a few to face centre, none to lowest part of face centre; a very few post-centrally	Nil	Nil	Lesion ...	Nil ...	Nil
Cat 247	Junction of middle and lower third of nucleus lateralis	Pre- and post-crucial gyri; sigmoid and anterior part of lateralis	Nil	Nil	Nil ...	Lesion ...	Nil

Lesions of Nucle

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus ve tralis
Rh. 272	Dorsal border of ventral third of nucleus lateralis	Pre- and post-central gyri, precentral fibres only go to operculum; postcentral much fewer, stop opposite lower end of intra-parietal sulcus	Nil	Nil	Nil	Lesion. Nil	Nil ...

ntre median and nucleus arcuatus	Pulvinar	Nucleus ruber	Descending fibres to medulla and pons	Cord	Fibres elsewhere	Remarks
l ...	Nil ..	Nil	Nil	Nil	Nil	Needle introduced horizontally.
ateralis (B) *Middle Third.*						
l ..	Nil ...	Nil	Nil	Nil ...	Nil	Needle introduced horizontally.
l ...	Nil ...	Nil	Nil	Nil ...	Nil	Needle introduced horizontally.
l	Nil ...	Nil	Nil	Nil ...	Fibres to anterior colliculus; these are pallio-tectals cut across in their passage through nucleus ventralis	Needle introduced horizontally.
ateralis (C) *Ventral Third.*						
il ...	Nil	Nil	Nil	Nil	Nil	Needle introduced horizontally.

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus v tralis
Rh. 216	In nucleus arcuatus and cutting the lower edge of centre median	Nil	Nil	Fibres break up here	Fine fibres break up in upper part of lateral nucleus. In addition some of superior; peduncular fibres can be seen degenerated	Fine fibres break up here	Fine fil break here
Rh. 218	In nucleus arcuatus cutting lower edge of centre median	Nil	Nil . ..	Fine fibres break up here	Fine fibres break up here	Fine fibres break up here	Fine fib break here
Rh. ·275	In centre median ...	Nil	Nil	Nil	Fine fibres break up here	Nil	Nil ..
Rh. 267	In centre median and on border between it and nucleus lateralis (ventral portion)	Nil	Nil	Nil	Some fibres enter nucleus lateralis and break up in its inner half, extending over an area corresponding to the sectional area of lesion	Fine fibres break up here	Fine fib break here
Rh. 87	In lower part of centre median and nucleus arcuatus, partly in ventralis and cutting Meynert's bundle	Indeterminate	Nil	Fine fibres break up here coming up from lesion	Fibres go into posterior ventral fourth and into ventral border of superior nucleus and pulvinar	Fine fibres break up here	Lesion; fi fibres bre up here

Lesions

Rh. 203	In the trunk of median fillet posteriorly where it comes around the nucleus ruber and extends to beginning of corpus Luysii. In breadth nowhere exceeds the breadth of fillet	Indeterminate ...	Nil	Nil	A few fillet fibres end here	Most of the fillet fibres break up here	Nil ...

Nucleus Arcuatus.

tre median c nucleus rcuatus	Pulvinar	Nucleus ruber	Descending fibres to medulla and pons	Cord	Fibres elsewhere	Remarks
ion ..	Fibres break up here	Nil	Nil	Nil . . .	Nil	Needle introduced horizontally.
ion . .	Fibres to pulvinar	Nil	Nil	Nil	Nil	Needle introduced horizontally.
ion fibres n down to nu us arcu as	Nil ..	Nil	Nil	Nil	Nil	Needle introduced horizontally.
ion . .	Nil ...	Nil	Nil	Nil	Nil	Needle introduced horizontally.
sion; res pass rough cleus cuatus d end in ticular ea	A few fibres end here	Nil	The posterior longitudinal bundle is degenerated in part. Fibres are seen ending in nuclei of cranial nerves (V, VI, VIII, X)	Nil	Fibres run from lesion in Forel's field and end in globus pallidus; Meynert's bundle degenerated to interpeduncular ganglion	Needle introduced vertically.

pothalamus.

... ...	Nil	Fine fibres go to capsule of lower part of homolateral nucleus and to the contralateral nucleus ruber	Nil	Nil	Nil	Needle introduced horizontally and passed along the course of the median fillet. On account of bruising of the cortex in removing the brain the cortex was not examined; however, the internal capsule was clear and contained no degeneration.

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus tral
Rh. 207	Lower part of ventral nucleus and upper border of Forel's field anteriorly cuts the lower end of the fan-shaped distribution of the fillet	Operculum ? ...	Nil	Nil	Fillet fibres break up here	Fillet fibres break up here	A few fi end her

Lesion

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus tral
Rh. 236	Large lesion, central focus of which is in posterior limb and outer part of genu of internal capsule, also destroys anterior superior mesial third of the lateral nucleus; edge of anterior nucleus, of nucleus medius, of caudate and tænia thalami	A few very fine fibres to frontal gyri; coarser fibres to pre- and post-central gyri; fewer and finer fibres to post-central gyrus	A few fine fibres	A few fine fibres break up herein	Large bundles of fine fibres entering anterior and middle third (fronto-thalamic lenticulo-thalamic)	Some fibres in this nucleus in both hemispheres	Some fi break herein
Rh. 258	In internal capsule in front of and to outer side of nucleus anterior needle track passes through dorsal part of nucleus lateralis	Fine fibres to inferior and middle frontal gyri; a few coarser fibres from needle track to pre- and post-central gyri	Nil	Nil	Lesion ...	Nil	Nil ...
Rh. 251	Anterior limit is in most basal part of capsule just behind the genu. Posterior limit in Forel's field on a level with centre of corpora mammillaria	Nil	Nil	Nil	Fillet fibres break up herein	Fillet fibres break up here	Fine fibr ventral cleus break herein

Lesions

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus tral
Cat 91	Pulvinar and anterior colliculus	Indeterminate .	Indeterminate ...	Indeterminate	Indeterminate	Indeterminate	Indeter nate

re median nucleus cuatus	Pulvinar	Nucleus ruber	Descending fibres to medulla and pons	Cord	Fibres elsewhere	Remarks
v to cen- median; t seem end in leus ar- tus	Nil ..	A few break up here	Nil	Nil	Medium-sized fibres to globus pallidus	Needle introduced horizontally.

sula Interna

.. ...	Nil ...	Nil	Nil	Pyramidal degeneration	A few fine fibres to caudate and globus pallidus and middle zone of nucleus lenticularis. Fibres in Schultze's bundle	This lesion was made large with the idea of producing symptoms. It was, however, anatomically not very satisfactory because of its size and the capsular injury. Needle introduced horizontally.
... ..	Nil	Nil	Nil	Pyramidal fibres from lesion of capsule	Nil	Needle introduced horizontally.
..	Nil ...	Some fibres from needle track break up here. Fibres from lesion end in anterior capsule of nucleus ruber	Tecto-pontine fibres from needle track	Nil	Some fine fibres to tuber cinereum and to locus niger, another group cross the capsule, and break up in the globus pallidus	Needle introduced horizontally. Needle track passed through the fillet.

sencephalon.

e termi- e	Lesion ...	Nil	Tecto-pontine fibres; most dorsal fibres of posterior longitudinal bundle; these latter disappear before they reach the cord	Nil	Fibres cross posterior commissure and break up in reticular substance of opposite side in plane of base of the aqueduct of Sylvius	As needle was introduced vertically the cortical injury permits only of a positive opinion being expressed on the descending fibres.

No.	Lesion	Cortical fibres	Nucleus anterior	Nucleus medius	Nucleus lateralis	Nucleus lateralis, ventral portion	Nucleus trali
Rh. 211	In upper nucleus of nucleus ruber	Nil	Nil . ..	Nil	Nil	Fine fibres break up herein and in ventral part on the contra-lateral side	Nil . .
Rh. 273	Anterior part of anterior colliculus and cutting the anterior brachium and a few of the dorsal terminal fibres of the superior cerebellar peduncle	Nil	Nil	A few medium-sized fibres run here	Fine fibres break up herein	Nil	Nil ...

Further, the rubro-bulbar tract now described occupies the region through which also pass the fibres of the tractus recurrens of the superior cerebellar peduncle (Thomas [152], Van Gehuchten [63], Horsley and Clarke), and it also ceases in the nucleus centralis reticularis and the nucleus magno-cellularis bulbi. The tract in question may have been injured by the lesion in Ernst's [47] experiment, but at a point below the nucleus ruber.

In leaving the discussion of lesions of the hypothalamic region it is necessary to draw attention to the fact that, though the experiments reveal a close association between the nucleus ruber and the globus pallidus, there is, as regards downward directed paths, no evidence of a direct connection between the thalamic nuclei (for instance, the nucleus lateralis) and the nucleus ruber. It is evident the whole important question of the downward or efferent connection of the thalamus and nucleus ruber requires a complete re-examination and research.

STIMULATION EXPERIMENTS.

In the stimulation experiments the double needles mentioned in the section on "Method and Technique" were used throughout. In employing these needles a bipolar excitation could be applied to an

median nucleus atus	Pulvinar	Nucleus ruber	Descending fibres to medulla and pons	Cord	Fibres elsewhere	Remarks
w fine es end in	Nil	Lesion ...	Monakow's bundle consisting of rubrospinal and rubrobulbar; of the former only two or three fibres. Another tract starts from anterior end of nucleus ruber, decussates in Forel's commissure and passes down just outside of medullary sheet, and apparently ends in nucleus magno-cellularis; a few fibres go down posterior longitudinal bundle	Fibres in anterior column from the posterior longitudinal bundle fibres	A very few fibres of medium size go into globus pallidus	Needle introduced horizontally.
lium- d fibres ak up upper d pos- or part entre lian	Nil	Nil	Tecto-ponto-bulbar tract. These fibres run down the same side near the median line and break up in the substance of the pons	Nil	Pallio-tectal fibres	Needle introduced horizontally.

extremely small area, and thus the disconcerting and disturbing effects of unipolar stimulation were avoided. This point has been dwelt on in the foregoing pages, and at considerable length in Horsley and Clarke's article on the cerebellum in *Brain*, 1908. In order to obtain the most precise results the smallest double needles we had were used, and these measured on the average 1·5 mm. in diameter at the widest part of the collar.

The current was obtained from an induction coil which was supplied by two dry-cell batteries. The secondary coil was furnished with the Kronecker scale which, though it does not indicate the actual quantity of current used, shows the relative proportionate excitation—*i.e.*, if the coil is at 200 on the scale, and then is advanced to 400, the stimulus obtained at the latter position is twice that obtained at the first. The average strength of current used varied from 100-500 Kronecker scale divisions, and if any region was found to be inexcitable, this was further tested by raising the intensity of the stimulation until "spread" evoked a response from neighbouring structures.

In this respect, as in all excitation experiments, the mode of response clearly indicated what was the real local effect of the stimulus.

In the procedure of these experiments, which were all carried out on *Macacus rhesus*, the monkey—thoroughly anæsthetized—was placed in Clarke's instrument, and after the skull and dura had been opened the double needle was introduced usually 2 mm. at a time. Every 2 mm. a stimulation was made and the resulting phenomena recorded.

If the nerve centres did not show signs of fatigue the entire thalamus was thus stimulated. The very slight shock attending such an experiment is evidenced by the fact that good results may be obtained for two or three hours if the body temperature is not allowed to fall and the brain kept warm by a constant stream of hot water. There is no apparent change in the electrical excitability of the brain. Furthermore, the passage of these small needles produces such a slight injury to the cells that bringing back the needle to a point stimulated some time previously, and stimulating there again, yields the same results as the first time. As soon as the experiment was completed the monkey was killed and the brain injected *in situ* with formol-Müller. The inter-aural line was then marked by boring a hole through the external auditory meati and a rod dipped in carbon and oil passed through to mark the inter-aural line (*see* fig. 76, Slab 1). Other points were then marked on the cortical surfaces to establish the inter-aural plane and the brain was removed. The brain was then cut through the inter-aural plane and then further cut up into 2 mm. slabs on Clarke's macrotome.

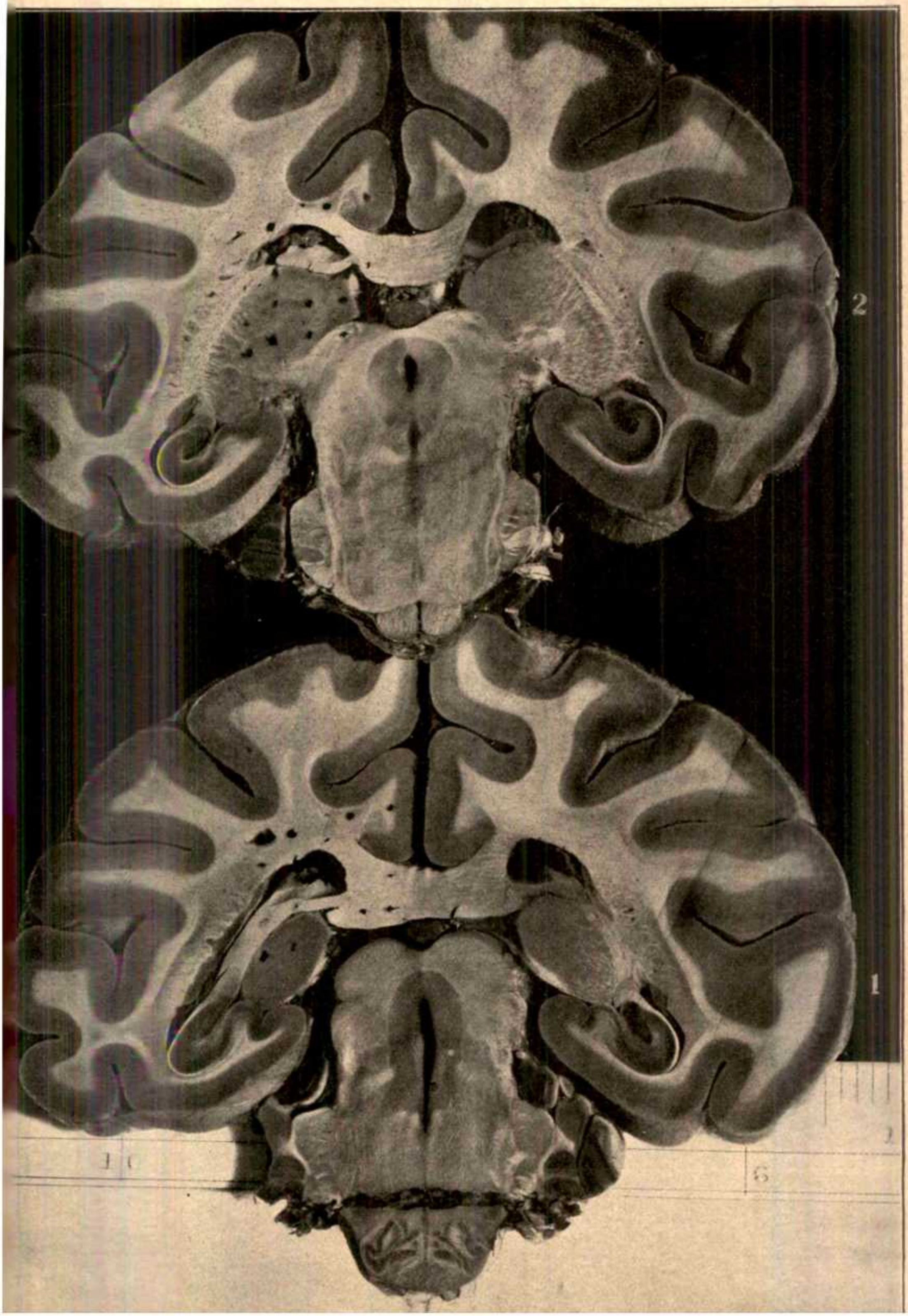
The exact points at which the stimuli were applied could then be determined, since, as can be seen on figs. 76 *et seq.*, the fine needle track examined immediately after death in the fixed brain is obvious as a small black point in frontal section. As stated in Horsley and Clarke's [75] paper, fixed points for localization purposes were also made as required by switching off the faradic current, and marking the spot excited by a small electrolysis.

The collective results of the experiments can best be stated, in so far as they followed the excitation of the various nuclei or fibres passing through them.

Nucleus Anterior.

This region is evidently extremely inexcitable, for even powerful currents produced no effect whatsoever. When the edges of the nucleus are reached, however, the same or similar symptoms may be observed as arise from regions adjacent to the anterior nucleus.

igs. 76-77. *Stimulation Experiment.* *Macacus rhesus.*—As seen in Slab 1, the inter-aural line passes through rapezial region. The brain was divided into slabs 2 mm. thick, numbered 1 to 8, but was not cut quite vertically, e the upper (thalamic) portion of Slab 2 is actually in the inter-aural or zero plane. The divisions of the scale n are millimetres; the magnification therefore about 2·5. The needle tracks are visible as dots, but the rtion of the brain after removal from the skull alters somewhat their 2 mm. relation to each other.



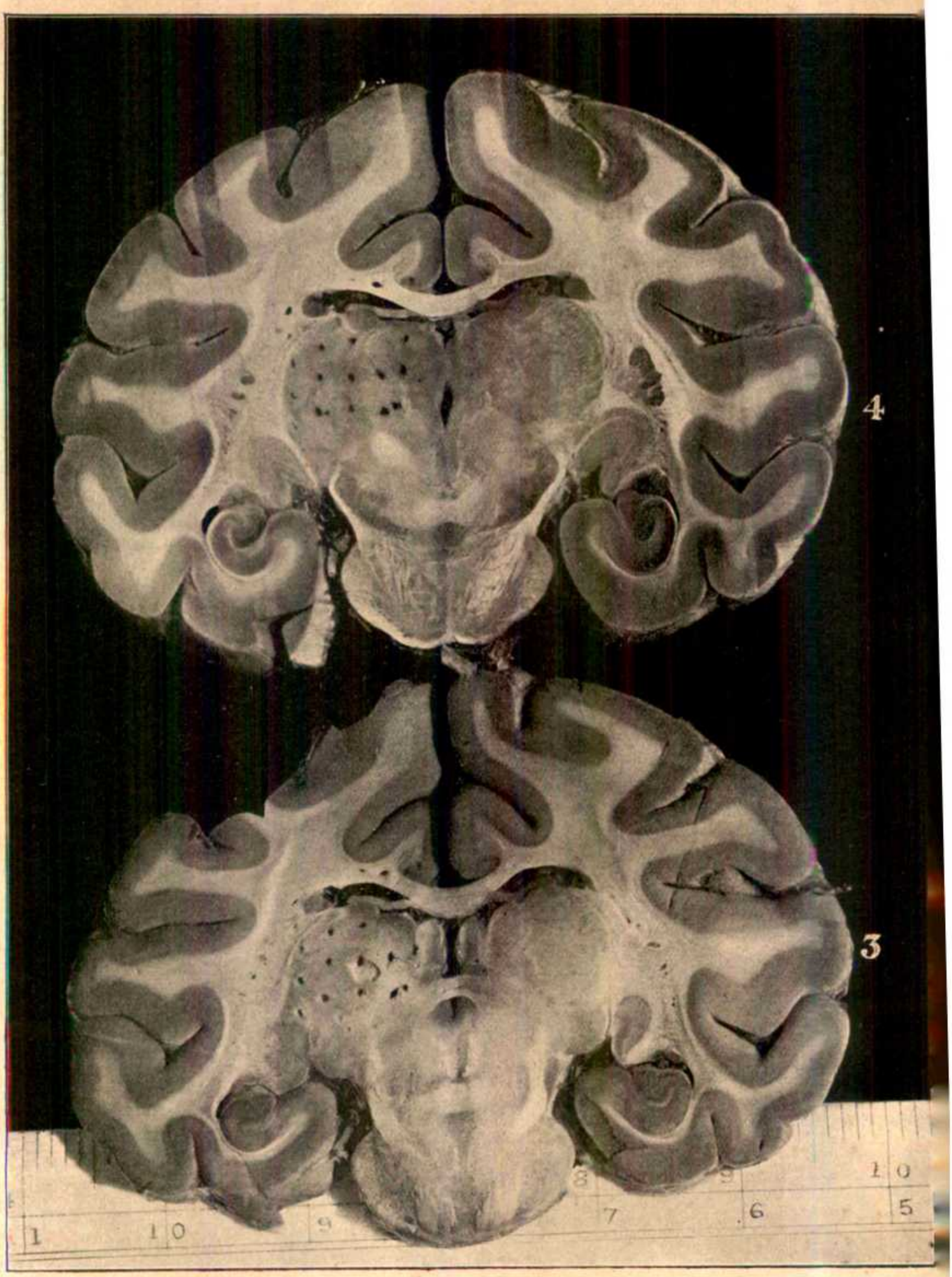


FIG. 77.

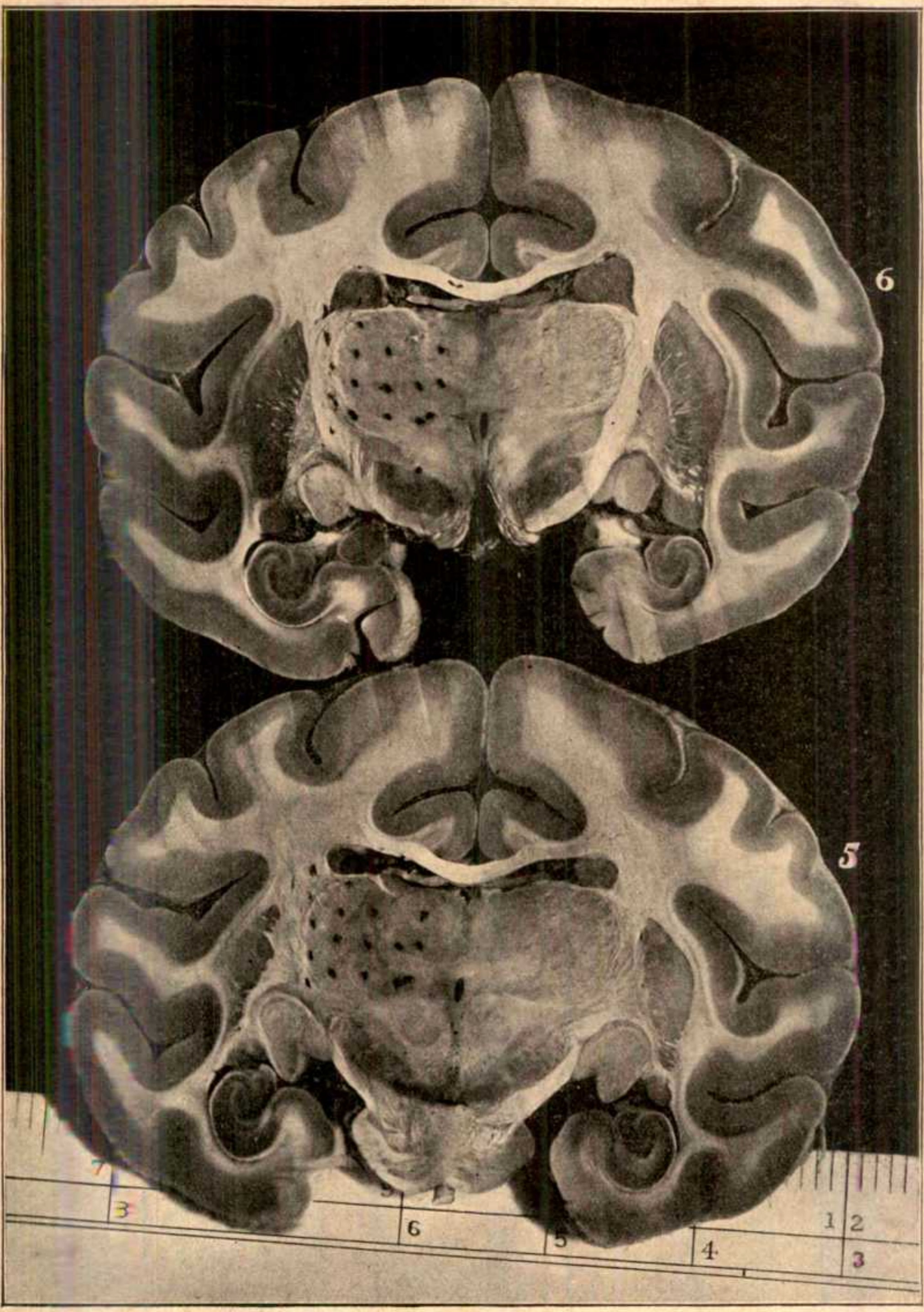


FIG. 78.

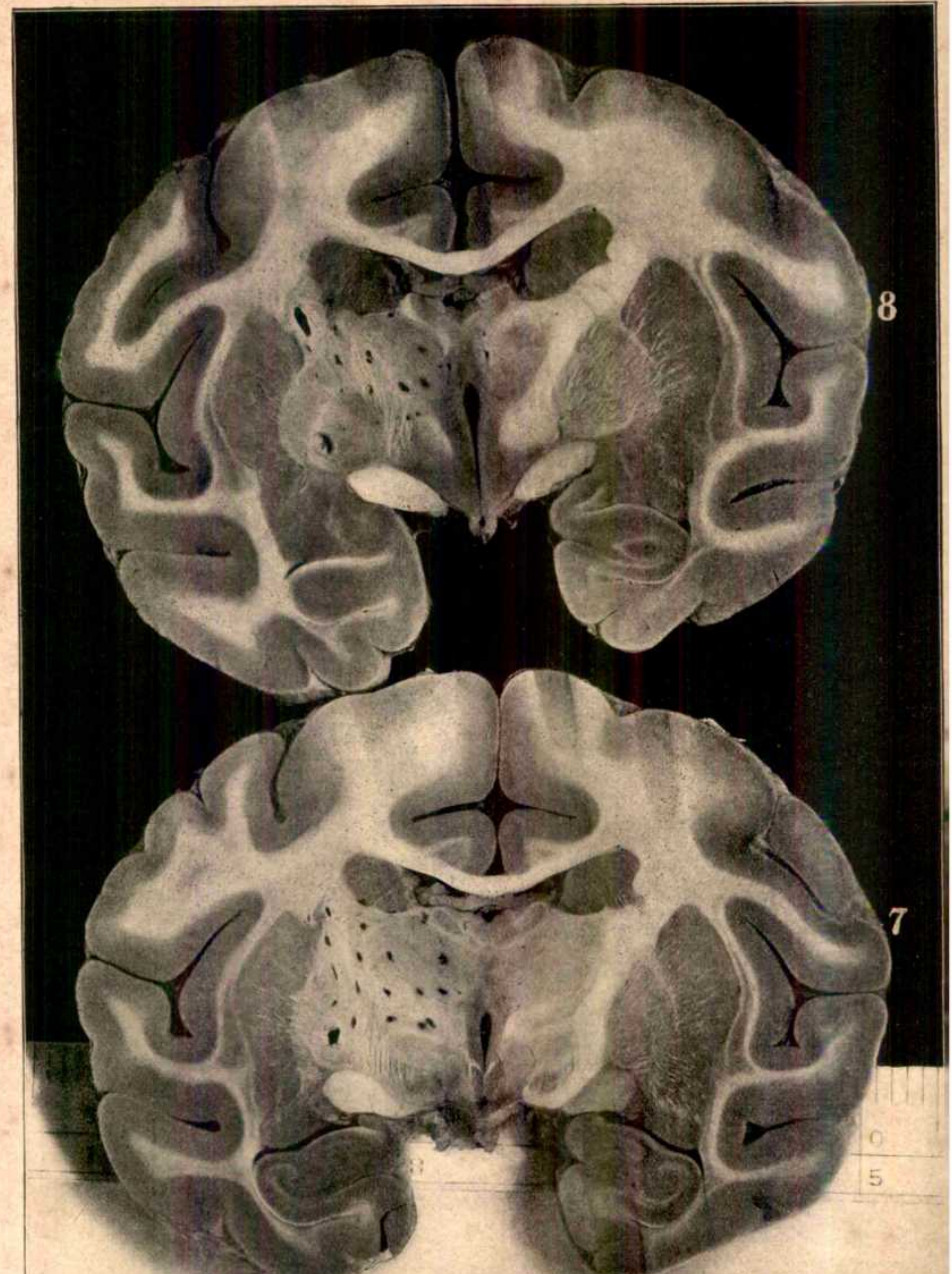


FIG. 79.

Nucleus Medius.

(*a*) The dorsal part of the nucleus, that lying directly beneath the tænia thalami, is inexcitable. When the central portions of the nucleus were stimulated, eye-movements were obtained, and these, with but occasional exceptions, consisted in the eyes being fixed with their visual axes parallel and parallel to the median sagittal plane.

Together with this there was at times dilatation of the pupils, but this was not a constant phenomenon. The appearance of the animal during excitation of the nucleus was that of attention. These movements were characterized by their comparatively slow onset, and contrast notably with ocular movements derived from any other part of the central nervous system in which the eye-muscles are represented.

(*b*) The ventral part of the nucleus is very interesting in its response to stimulation, inasmuch as, as soon as the needle advanced to and beyond the plane limiting the massa intermedia (commissura mollis) posteriorly, the excitation evoked a moderate deviation of the head and eyes to the opposite side and flexion of the opposite fore-limb. This is obviously the commencement of progression movement, as will be described by Horsley and Clarke, resulting from excitation of the fibres of the superior cerebellar peduncle (*cf.* also Thiele). These fibres have been found by the authors[1] just named to branch beneath and in the nucleus medius—*i.e.*, in the lamina interna.

Nucleus Lateralis.

Excitation of the nucleus lateralis has given constant results, but the interpretation of the effects observed requires careful consideration and merits further minute investigations in animals on whom exclusion experiments have previously been performed—*i.e.*, removal of the corresponding areas of the excitable cortex with establishment of pyramidal degeneration, &c. The present communications, therefore, will consist of a brief categorical summary of the points noted, without discussion of the detail of the probable paths taken by the nerve impulses.

Part of the Nucleus lateralis excited.	*Movement observed.*
Posterior Third Dorsal half ...	Conjugate deviation of the eyes to the opposite side.
Posterior Third Ventral half ...	Eyes open, progression movement, extension of opposite fore-limb, later flexion of fore-limb of the same side.

---

[1] These fibres are probably the source of the locomotory movements observed by Thiele on excitation of the cut surface of the posterior part of the median nucleus [149].

Part of the Nucleus lateralis excited.	*Movement observed.*
Middle Third Dorsal half	Movement of the opposite limbs, flexion and extension.
Middle Third Ventral half	Progression movement, facial movements of the opposite side.
Anterior Third Dorsal half	Capsular movements.
Ventral half	Head turned to the opposite side, mouth open, mastication, face contralateral movement, tongue to the opposite side, flexion both fore-limbs, most on the opposite side. Flexion both hind limbs.

It is desirable, perhaps, to repeat here that these motor phenomena constitute no evidence of special centres in the thalamus for automatic or rhythmic movements, as has been assumed by some authors.

Centre Median and Nucleus Arcuatus.

The results obtained from this region are essentially compound—*i.e.*, a summation of those obtained by excitation of the median nucleus and of the superior cerebellar peduncular fibres.

Thus in all cases a combined movement of parallelism of the optic axes, with extension of the opposite fore-limb and flexion of the elbow of the same side, resulted from stimulation of the centre median and nucleus arcuatus.

Pulvinar.

Excitation of the pulvinar invariably produced conjugate deviation of the eyes to the opposite side, with dilatation of the pupils.

In time the rate of this movement was between that obtained from the colliculus anterior and that from the median nucleus (the slowest, *v. supra*).

A special point in reference to the pupil must be mentioned here. In a constant series of observations a very sharp, quick contraction of the pupils was obtained from a spot where the outer part of the posterior commissure crosses behind Meynert's bundle. This is a direct effect of the excitation communicated to the third nucleus, and is very striking in view of the widespread representation of dilatation all round this point.

SYMPTOMS.

The symptoms observed in each animal the subject of an experimental lesion must be divided according to the site of the lesion—cortical or thalamic.

(a) Symptoms following lesions of the cortex cerebri.

Since the researches of Munk and Goltz attention has been sufficiently given to the symptoms which immediately follow the production of a destructive lesion due to the disturbance of an operation, and those which permanently remain as consequences of the lesion itself. In every case the transient symptoms have been noted, but need not be referred to now.

As regards permanent changes, little has been observed except in lesions of the sensori-motor area. Further, whereas the cat responds readily to all forms of sensory stimulation, the monkey is exceedingly indifferent, and the accurate estimation of sensory changes in this animal presents many difficulties. However, in all cases each animal was systematically and repeatedly examined as to motor power, co-ordination, hemianopsia, æsthesia to touch, cold, heat, pin-prick, and in the monkey also astereognosis.

(1) *Cats.*

(a) Sensori-motor region (*see* Table I).

Lesions of the cruciate region were followed by loss of sense of position, thermo-anæsthesia to cold and paresis of special (voluntary) movements in the contralateral limbs.

(b) Parieto-temporal regions (*see* Table I).

From the small lesions of the other parts of the cortex no symptoms were distinguishable.

(2) *Monkeys* (*Macacus rhesus*).

In this animal the lesions were confined to the central gyri.

(a) Gyrus precentralis (*see* Table I).

The monkeys with precentral lesions showed paresis of the extremity corresponding to the cortical area destroyed, and uncertainty in use of the affected limb when jumping and climbing free in a large cage.

No sensory changes could be detected. The knee-jerk of the opposite side was increased except when at a later stage contracture commenced. It was then diminished.

(b) Gyrus postcentralis (*see* Table I).

The monkeys with postcentral lesions were apparently quite normal. In their free movements and to every test their sensory and motor power appeared unaltered. In one case of a postcentral lesion there were symptoms of paresis and uncertainty, and this therefore appeared to constitute an exception. The serial sections of the brain, however, showed a thrombotic lesion, low down in the corona radiata, that had cut off precentral fibres. The experiment, therefore, confirms the facts given above in differentiation between the two central gyri.

(*c*) Symptoms following lesions of the optic thalamus.

Since the earliest investigations on the thalamus down to the most recent work by Roussy two cardinal symptoms have always been recorded as characteristic of such a lesion. (1) Mouvement de manège; (2) hemianopsia.

All observers, as far as could be discovered in the literature, have noted those two symptoms, with the sole exception of Nothnagel. It must seem very surprising, therefore, that in the present research twenty-two Rhesus monkeys and four cats, all with thalamic lesions, not one showed the symptom of mouvement de manège, or anything at all suggesting it, and only four showed ocular symptoms.

Rh. 275 and Cat 91 both exhibited right-sided hemianopsia. Rh. 236 noticed threatening movements less on the right side than the left, but showed no signs of hemianopsia when taking his food or with currants strewn about the floor. Cat 95's pupils reacted sluggishly to light, but there was no evidence of hemianopsia.

The explanation of such absence of symptoms must be that the small lesions of the present research only destroyed small parts of the nuclei, and consequently that sensory representation of any given region could only have been slightly affected. That this is probably correct is shown by the fact that in one case where a considerable injury was done to the pulvinar, hemianopsia resulted, and that in the case (Rh. 211) where the lesion was situated in the nucleus ruber the opposite ear was drawn to the shoulder.

Any thalamic syndrome must consist of an arrangement of disordered function of those senses whose special thalamic regions are destroyed, and of those tracts—*e.g.*, the internal capsule, optic tract, &c.—which lie near.

SUMMARY.

(1) The hypothalamus is essentially distinct from the thalamus.

(2) The hypothalamus is closely connected with the globus pallidus.

(3) The thalamus must be regarded as consisting of an inner and outer division, of which the inner includes the nucleus anterior and nucleus medius.

4) The inner division is in association with the nucleus caudatus and with the rhinencephalon.

5) The outer division is the end station of the fillet and of the superior cerebellar peduncle. It is closely connected with the Rolandic region of the pallium.

6) From the results of excitation experiments, as well as the anatomical facts deduced from very localized lesions, the inner and outer divisions of the thalamus appear to be in the main relatively independent organizations.

7) The precentral especially and postcentral pallium (*Macacus rhesus*) and the excito-motor area in the cat are connected by pyramidal fibres and collaterals with the middle and ventral thirds of the nucleus lateralis.

8) The temporal pallium (cat) is connected by projection fibres and collaterals with the postero-ventral region of the nucleus lateralis, the corpus geniculatum mediale and the pons varolii.

9) The thalami-cortical fibres connecting the nucleus lateralis with the pallium are arranged dorso-ventrally, so that those for the representation of the face are ventral to those for the limbs.

10) The general direction of the large majority of axones, whether originating in a thalamic nucleus or passing through the thalamus, is outwards and dorsalwards.

11) The precentral pallial area for the representation of the movements of the limbs is not connected by pallio-tectal fibres with the colliculi.

The figures illustrating this paper are furnished to show the size and position of the lesions on which the statements in the text and in Table II are founded.

The lettering throughout is the same, as follows :—

AC = commissura anterior.
AN = nucleus anterior.
BA = brachium anticum.
BP = brachium posticum.
BT = fasciculus temporo-pontalis (Türck's bundle).
C = crus.
CA* = colliculus anterior (corpus quadrigeminum superior).

* In Rh. 251 CA is by error used to denote a corpus albicantium v. mammillare.

CCA = corpus callosum.
CG = griseum centrale.
CGE = corpus geniculatum externum.
CI = capsula interna.
CL = corpus hypothalamicum (Luysii).
CM = *centre médian.* Luys.
CN = nucleus caudatus.
CP = colliculus posterior (corpus quadrigeminum inferior)
CR = corona radiata.
d = degeneration.
f = fornix longus.
FL = fillet (lemniscus medialis).
FOL = tractus areae forellii—lenticularis (see text)
FOR or FO = field of Forel.
fd = degeneration.
fdm = degeneration of pyramidal system.
GP = globus pallidus.
GPoC = gyrus postcentralis.
GPrC = gyrus precentralis.
L or l = lesion.
Li = lamina interna.
LL = lattice layer.
LN = nucleus lateralis.
ln = locus niger.
LPP = locus perforatus posticus.
LV = ventriculus lateralis.
M = fasciculus retroflexus (Meynert's bundle).
MI = massa intermedia (commissura mollis).
MN or NM = nucleus medius.
N.II = nervus opticus.
N.III = nervus tertius v. oculo motorius.
NA = nucleus arcuatus.
NL = nucleus lenticularis.
NN = nervus quartus.
n = needle track.
PCO = operculum precentralis.
PT = tractus palliotectalis.
PU or P = pulvinar.
RGP = tractus rubro-globus-pallidus.
Sc = stratum subcaudatum (see text)
SP = pedunculus cerebelli superior.
Sp = splenium.
T = tænia thalami.
TB = tractus tecto-bulbaris.
V.III = ventriculus tertius.
VA = fasciculus Vicq. d'Azyri.
VN or NV = nucleus ventralis.

BIBLIOGRAPHY.

[1] D'ABUNDO. "Contributo allo studio del sistema nervoso," *Neurol. Centralbl.*, 1898, Bd. xvii, S. 68.

[2] ARNOLD. "Handbuch der Anatomie," Zürich, 1838-42, 1851.

[3] *Idem.* "Anatomie des Gehirns und Ruckenmark," 1838.

[4] ASHER. *Corresp. Blatt. f. schweiz. Aerz.*, 1901, Bd. xxxi., S. 461.

[5] BALFOUR. "Comparative Embryology," London, 1881.

[6] BAGINSKY. *Virchow's Archiv.*, 1886, Bd. cv., S. 28, and 1890, Bd. cxix., S. 81.

[7] BECHTEREW. *Neurol. Centralbl.*, 1906, Bd. xxv., S. 546.

[8] *Idem.* "Über die sensible und motorische Rolle des Sehhügels," *Monatschr. f. Psych. und Neurol.*, 1905, Bd. xvii., S. 224.

[9] *Idem.* *Virchow's Archiv.*, 1887, Bd. cx., S. 102.

[10] *Idem.* *Neurol. Centralbl.*, 1890, Bd. ix., S. 642.

[11] *Idem*, 1894, Bd. xiii., S. 252.

[12] *Idem*, 1897, Bd. xvi., S. 146.

[13] BECHTEREW and MISLAWSKI, "Uber die Hirncentren der Thränenabsonderung," *Neurol. Centralbl.*, 1891, Bd. x., S. 481.

[14] *Idem*, 1888, Bd. vii., S. 505.

[15] *Idem.* "Über die Innervation des Darmes," *Arch. f. Anat. u. Physiol.* (Phys. Abth.), 1889, Suppl. Bd., S. 243.

[16] *Idem.* "Über die Innervation des Magens," *Neurol. Centralbl.*, 1890, Bd. ix., S. 173.

[17] *Idem.* *Archiv. f. Anat. u. Physiol.* (Phys. Abth.), 1891, S. 380.

[18] BEEVOR. *Phil. Trans. of the Roy. Soc.*, B., 1891, vol. clxxxii., p. 135.

[19] *Idem*, vol. cc., p. 108.

[20] *Idem* and HORSLEY. "On Pallio-tectal Fibres," *Brain*, 1902, vol. xxv., p. 436.

[21] BENAKY. "Contribution à l'étude des fonctions de la couche optique," *Archiv. de Neurol.*, 1904, vol. xviii., p. 385.

[22] BETHE. "Anatomie und Physiologie des Nervensystems," Leipzig, 1903.

[23] BICKEL. "Physiologie des Gehirns," *Pflüger's Archiv.*, 1898, Bd. lxxii., S. 190.

[24] BOYCE. *Phil. Trans. of the Roy. Soc.*, B., 1895, vol. clxxxvi., pt. i., p. 321.

[25] BRUCE. "Tracts of Mid and Hind Brain," Edinburgh, 1892.

[26] BURDACH. "Bau und Leben des Gehirns," Leipzig, 1819.

[27] CAJAL. "Las fibras nerviosas de origen cerebral del talamo optico," Fourteenth International Congress of Medicine, Madrid, 1903, Sect. Anat., p. 36.

[28] *Idem.* "Textura del sistema nervioso," 1902.

[29] CAMPBELL. "Histological Studies on the Localization of Cerebral Function," Cambridge, 1905.

[30] CARPENTER. "Principles of Physiology," London, 1851.

[31] CARVILLE and DURET. "Sur les fonctions des hemisphères cérébraux." *Archiv. de Physiol.*, 1875, vol. vii., p. 352.

[32] CASTELLINO. "Ueber die Function des Thalamus," *Wien. med. Wochenschr.*, 1895, Bd. xlv., S. 1395.

[33] COLLIER and BUZZARD. "Descending Mesencephalic Tracts, &c.," *Brain*, 1901, vol. xxiv., p. 177.

[34] CONTJEAN et TISSOT. "Sur l'excitation des centres cérébraux," *Compt Rend. Soc. de Biol.*, 1897, 10me sér., vol. iv., p. 113.

[35] COUTY. "Lésions du cerveau chez le chien et le singe," *Compt. Rend. Soc. de Biol.*, 1881, 7me sér., vol. iii., p. 75.

[36] DANA. "Functions of the Corpora Striata," *Journ. of Nerv. and Ment. Dis.*, 1908, vol. xxxv., p. 65.

[37] DARKSCHEWITSCH. *Neurol. Centralbl.*, 1885, Bd. iv., S. 100.

[38] *Idem* and PRIBYTKOW. *Neurol. Centralbl.*, 1891, Bd. x., S. 417.

[39] Deiters. "Untersuchungen über Gehirn u. Rückenmark," Braunschweig, 1865.

[40] Dejerine. "Anatomie des centres nerveux," Paris, 1895, 1901.

[41] *Idem* and Long. "Sur les connexions de la couche optique avec la corticalité cérébrale," *Compt. Rend. Soc. de Biol.*, 1898, 10me sér., vol. v., p. 1131.

[42] Dupuy. "Leçons sur les fonctions du cerveau," *Soc. de Biol.*, 1884.

[43] Edinger. *Neurol. Centralbl.*, 1893, Bd. xii., S. 327. *Archiv. f. Psychiat.*, 1895, Bd. xxvii., S. 950.

[44] *Idem.* "Vom Verlauf der Gefühlsbahnen," *Deutsch. med. Wochenschr.*, 1890, Bd. xvi., S. 421.

[45] *Idem.* "Vorlesungen über das Centralnervensystem," Bd. i. and ii., Leipzig, 1904, 1908.

[46] Eisenlohr. "Zur Hirnlocalisation," *Deutsch. Zeitsch. f. Nervenheilk.*, 1893, Bd. iii., S. 260.

[47] Ernst. "On the Descending Tracts from the Thalamus and Anterior Corpora Quadrigemina." Dissertation (Russian), St. Petersburg, 1902.

[48] Ferrier. "Functions of the Brain," London, Second Edition, 1886.

[49] *Idem* and Turner. *Phil. Trans. of the Roy. Soc.*, B., 1898, vol. cxc., p. 1.

[50] Flatau and Jacobson. "Vergleichende Anatomie," Berlin, 1899.

[51] Flechsig. "Leitungsbahnen in Gehirn," 1876.

[52] *Idem. Neurol. Centralbl.*, 1897, Bd. xvi., S. 290.

[53] *Idem. Arch. f. Anat. u. Physiol.* (Anat. Abth.), 1881, S. 12.

[54] Flourens. "Recherches expérimentales sur les fonctions du système nerveux," Paris, 1842.

[55] Forel. "Untersuchungen über die Haubenregion und ihre oberen Verknüpfungen, &c." *Arch. f. Psych.*, 1877, Bd. vii., S. 393.

[56] *Idem.* "Einige Hirnanatomische Ergebnisse," *Arch. f. Psych.*, 1887, Bd. xviii., S. 162.

[57] Foville. "Traité de l'Anatomie du système nerveux," Paris, 1844.

[58] Fournié. "Recherches sur les fonctions du cerveau," Paris, 1873.

[59] France. *Phil. Trans. of the Roy. Soc.*, B., 1889, vol. clxxx., p. 331.

[60] Froin. "Etude des localisations dans les noyaux gris," *Compt. Rend. Soc. de Biol.*, 1903, vol. lv., p. 1657.

[61] Ganser. "Anatomische Studie über das Gehirn des Maulwurfs," *Morphol. Jahr.*, Bd. vii., S. 591.

[62] Gehuchten, van. "L'anatomie du système nerveux de l'homme."

[63] *Idem.* "Névraxe," 1903, vol. v., p. 263; 1905, vol. vii., p. 29.

[64] Gentes and Aubaret. "Connexion de la voie optique," *Compt. Rend. Soc. de Biol.*, 1902, vol. liv., p. 1283.

[65] Goltz. *Pflüger's Archiv.*, 1881, Bd. xxvi., S. 1; 1882, Bd., xxviii., S. 579; and 1888, Bd. xxxiv., S. 451.

[66] Gowers. "Case with Lesion in Optic Thalamus," *Journ. Neurol.*, 1892.

[67] Gudden. *Arch. f. Psych.*, 1870, Bd. ii., S. 693.

[68] Hale White. "Rise of Temperature from Lesions of Thalamus," *Brit. Med Journ.*, 1889, vol. i., p. 1401.

[69] Hamilton. *Proc. Roy. Soc.*, 1884, vol. xxxvi., p. 349.

[70] Held. *Arch. f. Anat. u. Physiol.*, 1891 (Anat. Abth.), S. 271, and 1893 (Anat. Abth.), S. 201.

[71] Henle. "Anatomie des Menschens," Braunschweig, 1868.

[72] Hitzig. *Arch. f. Psych.*, 1903, Bd. xxxvi., S. 1.

[73] Horsley and Schäfer. *Proc. Roy. Soc.*, 1884, vol. xxxvi., p. 437.

[74] *Idem*, 1885, vol. xxxix., p. 404.

[75] *Idem* and Clarke. *Brain*, 1908, vol. xxxi., p. 1.

[76] Hosel. "Ueber die Markreifung der Körperfühlsphere," *Arch. f. Psych.*, 1905, Bd. xxxix., S. 195.

[77] Irwanow. Dissertation, St. Petersburg, 1899.

[78] Jackson. "Functions of Optic Thalamus," *Clinical Lectures and Reports of London Hospital*, 1866, vol. iii., p. 373.

[79] Jelgersma. *Neurol. Centralbl.*, 1895, Bd. xiv., p. 290.

[80] Kirchhoff. "Ein mimisches Centrum im medialen Kern des Sehhügels," *Arch. f. Psych.*, 1902, Bd. xxxv., S. 814.
[81] *Idem.* *Arch. f. Psych.*, 1897, Bd. xxix., S. 888.
[82] Kirilzew. *Neurol. Centralbl.*, 1891, Bd. x., S. 405.
[83] Kölliker. "Anatomie des Nervensystems."
[84] Kohnstamm and Quensel. *Neurol. Centralbl.*, 1908, Bd. xxvii., S. 242.
[85] Lafforgue. "Etude sur la lesion de la couche optique," Paris, 1877.
[86] Langley und Grünbaum. *Journ. Physiol.*, 1890, vol. xi., p. 606.
[87] Lenhossek. *Neurol. Centralbl.*, 1894, Bd. xiii., S. 412.
[88] Lewandowsky. "Die Functionen des Nervensystems," Jena, 1907.
[89] Lo Monaco. *Arch. Ital. de Biol.*, 1898, vol. xxx., p. 198.
[90] *Idem.* *Neurol. Centralbl.*, 1901, Bd. xx., S. 118.
[91] Long et Roussy. "Hémianesthésie cérébrale par lésion de la couche optique," *Revue Neurolog.*, 1906, vol. xiv., p. 1202.
[92] Longet. "Anatomie du système nerveux," 1846.
[93] Luciani. "Das Kleinhirn," Leipzig, 1893.
[94] Lussane and Lemoigne. *Arch de Physiol.*, 1877, vol. ix., p. 119.
[95] Luys. "Sur le système nerveux," Paris, 1865.
[96] Magendie. "Le système nerveux," Paris, 1841.
[97] Mann, G. "On the Optic Thalamus," *Brit. Med. Journ.*, 1905, vol. ii., p. 289.
[98] Marburg. "Atlas des menschlichen Gehirns," 1904.
[99] Marinesco. "Des connexions du corps strié," *Compt. Rend. Soc. de Biol.*, 1895, 10me sér., vol. ii., p. 77.
[100] *Idem.* *Semaine Méd.*, vol. xvi., 1896, p. 214.
[101] Marchi. "Sulla strutura dei thalami ottici," *Rivist. sper. di fren.*, 1884, vol. x., p. 329; *ibid*, 1886, vol. xii., p. 285.
[102] May, P. "Degenerations in Posterior Columns of Cord," *Rev. of Neurol. and Psych.*, 1905, vol. iii., p. 1.
[103] *Idem.* Referred to by Mann, G. (*see* above).
[104] Meynert. *Wien. med. Jahrsb.*, 1872, Bd. ii., S. 188.
[105] *Idem.* "Psychiatrie," Wien., 1884.
[106] Moeli und Marinesco. *Arch. f. Psych.*, 1892, Bd. xxiv., S. 655.
[107] Monakow, von. "Untersuchungen über den Thalamus opticus, &c.," *Arch. f. Psych.*, 1895, Bd. xxvii., S. 1, 386.
[108] *Idem.* *Arch. f. Psych.*, 1882, Bd. xii., S. 141, 535.
[109] Monakow. "Gehirnpathologie," "Nothnagel Specielle Pathologie und Therapie," Wien, 1897, Bd. ix., Th. 1.
[110] Mott. *Brain*, 1895, vol. xvii., p. 1.
[111] Munk. "Über die Funktionen des Grosshirns," Berlin, 1890.
[112] Münzer und Wiener. "Das Gehirn des Kaninchens," *Monatschr. f. Psych.*, 1902, Bd. xii., S. 241.
[113] Muratoff. *Neurol. Centralbl.*, 1893, Bd. xii., S. 714.
[114] Nissl. "Die Kerne des Thalamus beim Kaninchen," *Neurol. Centralbl.*, 1889, Bd. viii., S. 549.
[115] Nothnagel. "Untersuchungen am Gehirn," *Virchow's Archiv.*, 1874, Bd. lxii., S. 201.
[116] *Idem.* *Virchow's Archiv.*, 1873, Bd. lviii., S. 420.
[117] Obersteiner. "Nervöse Centralorgane," Leipzig u. Wien, 1901.
[118] Oppenheim. "Lehrbuch der Nervenkrankheiten," Berlin, 1905.
[119] Ott. "Vaso-tonic Centres in the Thalami," *Journ. of Nerv. and Ment. Dis.*, 1891, vol. xvii., p. 483.
[119a] Pagano. *Archives Italiennes de Biologie*, 1902, vol. xxxviii., p. 299.
[120] Pavlow. "Le Névraxe," 1900, vol. i., p. 235.
[121] Pick and Kahler. "Neue Untersuchungen uber das Nervensystem," *Arch. f. Psych.*, Bd. x., 1880, S. 179, 297.
[122] Preisig. *Journ. f. Psychol. u. Neurol.*, 1904, Bd. iii., S. 215.
[123] Probst. *Monatschr. f. Psych.*, 1900, Bd. vii., 387, and 1900, viii., 165.

[123A] *Idem*, 1901, Bd. x., S. 288.
[124] *Idem. Deutsch. Zeit. f. Nervenheilk.*, 1900, Bd. xvii., S. 141.
[125] *Idem.* 1898, Bd. xiii., S. 384.
[126] *Idem. Arch. f. Psych.*, 1900, Bd. xxxiii., S. 721.
[127] *Idem.* 1902, Bd. xxxv., S. 22.
[128] *Idem. Wien. klin. Wochenschr.*, 1902, Bd. xv., S. 932.
[129] *Idem. Sitz. der Wien. Akad. der Wissensch.*, 1905, Bd. cxiv., Abth. iii., S. 173.
[130] Prus. "Electrische Reizung des Thalamus opticus," *Wien. klin. Wochenschr.*, 1899, Bd. xii., S. 1199.
[131] Rethi. "Die Nervenwurzeln des Rachens, &c.," *Sitzb. d. Wien. Akad. d. Wissensch.*, 1893, Bd. cii., Abth. iii., S. 201.
[132] Retzius. "Das Gehirn des Menschens," Stockholm, 1896.
[133] Rothmann. "Versammlung der Deutsch. Naturforscher Gesellschaft," Dresden, 1907, Teil ii., 2 halfte, S. 205.
[134] Richter. "Zur Frage der Optischen Leitungsbahnen," *Arch. f. Psych.*, 1885, Bd. xvi., S. 639.
[135] Roussy. "La couche optique," Paris, 1907.
[136] Sachs. "Das Hemispherenmark," Breslau, 1892.
[137] Sachs. "Eine vergleichende anatomische Studie des Thalamus opticus der Säugetiere." Arb. aus d. Neurol. Instit. [Obersteiner], 1908, Bd. xvii., Hft. 2, S. 280.
[138] Schwalbe. "Lehrbuch der Neurologie," 1881.
[139] Schiff. "Experimentelle Physiologie," Florence, 1873.
[140] Sellier et Verger. "Lésions de la couche optique," *Compt. Rend. Soc. de Biol.*, 1898, 10me sér., vol. v., p. 522.
[141] *Idem*, 1903, 11me sér., vol. v., p. 485.
[142] *Idem.* "Recherches sur la physiol. de la couche optique," *Arch de Physiol.*, 1898, vol. xxx., p. 706.
[143] Serres et Loustan. "Anatomie du cerveau," Paris, 1824.
[143A] Simpson. *Proc. of the Physiolog. Soc.*, 1901-2, July 20, p. x.
[144] Sherrington and Grünbaum. *Proc. Roy. Soc.*, 1901, vol. lxix., p. 206.
[145] Soury. "Fonction du cerveau," 1892.
[146] Spitzka. *Neurol. Centralbl.*, 1885, Bd. iv., S. 246.
[147] Starr. *Journ. of Nerv. and Ment. Dis.*, 1884, vol. xi., p. 327.
[148] Tarasewitch. "Untersuchung über Thalamus, &c.," Arbeit. aus dem Neurol. Institut, Obersteiner, Wien, 1902, H. ix.
[149] Thiele. "Thalamus and Deiter's Nucleus," *Journ. of Physiol.*, 1905, vol. xxxii., p. 358.
[150] Trapieznikow. "Ueber die centrale Innervation des Schluckaktes. Diss. Inaug. an d. Petersbg. militärärztl. Academie." [Referat—*Jahresber. ü. Neurol. u. Psychiat.*, 1897.]
[151] Tschermak. "Ueber die aufsteigende Hinterstrang-Sbahn," *Arch. f. Anat. u. Physiol.*, 1898, S. 291.
[152] Thomas. *Journ. de Physiol.*, 1899, vol. i., p. 47.
[153] Veysierre. "Recherches sur l'hémianesthésie de cause cérébrale," *Thèse de Paris*, 1874.
[154] Vicq d'Azyr. "Recherche sur la structure de cerveau," Paris, 1805.
[155] Vogt. "Faisceau septo-thalamique," *Compt. Rend. Soc. de Biol.*, 1898, Sér. 10, v., p. 206.
[156] Wallenberg. "Ueber centrifugale Bahnen aus dem Sehhügel," *Neurol. Centralbl.*, 1901, Bd. xx., S. 50.
[157] Wernicke. "Gehirnkrankheiten," Kassel, 1881.
[158] Zacher. *Arch. f. Psych.*, 1891, Bd. xxii., S. 654.
[159] Ziehen. "Zur Physiol. der Infracorticalen Ganglien, &c.," *Arch. f. Psych.*, 1890, Bd. xxi., 863.
[160] *Idem.* "Anatomie des Nervensystems," Jena, 1902.
[161] *Idem.* "Untersuchungen über die Marsupialier," 1901.

MYOTONIA ATROPHICA.

BY FRED E. BATTEN AND H. P. GIBB.

INTRODUCTION.

THE object of this paper is to draw attention to a group of cases which present the rare association of muscular atrophy with a slow relaxation of muscles after voluntary contraction. The muscular atrophy has a distribution which is peculiar and corresponds to none of the well-known types of myopathy.

The characteristic features of the condition are weakness of the facial muscles (myopathic face), atrophy of the sterno-mastoids, atrophy of the vasti of the thighs and dorsiflexors of the feet, and a slow relaxation of certain muscles after contraction. The more forcible the contraction, the slower the relaxation.

We are aware that variations from the type have been described both in the distribution of the muscular atrophy and in the occurrence of the slow relaxation without atrophy, but considering the striking resemblances of our own cases, and those which we have collected from the literature, we think the type is worthy of recognition.

NAME.

This condition has been described under various names: Myotonia with muscular atrophy (Lannois). Muscular atrophy with the electrical reactions of Thomsen's disease (Lortat-Jacob et Thaon). Thomsen's disease with muscular atrophy (Nogues et Sirol). Atypical forms of Thomsen's disease (Pelz). Muscular atrophy with slow relaxation of muscles (nachdauernde Muskelkontractionen) (Kleist). Myotonia atrophica (Rossolimo and others).

We have adopted that of "myotonia atrophica," first used by Rossolimo, because it serves shortly to describe the salient features, and because it is the name under which several authors have since described this condition.

We are fully aware that objection may be raised to the term *myotonia*, since it is often used as synonymous with Thomsen's disease (myotonia congenita). Myotonia is, however, only a symptom, and, as such, its use is here justified. Objection to the term "atrophica" may be raised, since the atrophy does not always occur in the muscles which exhibit the myotonia; and it may further be urged that the name implies that the myotonia precedes the muscular atrophy, a sequence of events which does not occur in all cases.

Whilst recognizing these objections, we are of opinion that fuller recognition for this condition as an entity will be obtained by such a name as above suggested than by a lengthy title, which possibly may attain to slightly greater accuracy.

Case 1.—F. W., male, aged 40. This patient came under observation in 1906, and was shown before the Neurological Society as a "case of myopathy, exhibiting slow relaxation of muscles after muscular action." Since then there has been little alteration in his condition. He gave the following history —

In 1900 he noticed that his thighs were getting thin. The wasting caused him no inconvenience till 1903, when at times his knees would suddenly give way in walking, and he would fall. This difficulty in walking has persisted till the present time (1908). Recently he has had a stiffness about his legs, when he first starts walking, which passes off when he has gone a few yards. He complains, too, of a stiffness in the thighs when tired and of sharp shooting pains down both thighs and in small of the back. For the last few years he has noticed a steadily increasing weakness in his grip. The heavy work he is called on to do demands considerable strength, and he finds that he cannot grasp things as firmly as he could. He thinks, too, that of recent years there has been some general wasting of the forearms, especially along their ulnar borders.

For many years—he cannot say definitely how long—he has had difficulty in relaxing his grasp. He is certain the difficulty existed ten or twelve years ago. The difficulty passes off after repeated movements.

Since 1900—though here again he cannot date the onset very definitely—he has had difficulty in articulating his words when he first starts speaking. He has also had some stiffness about the jaws in chewing, and has noticed a tendency for his tongue to become stiff and "stick to the roof of his mouth" when he attempts to swallow. He has been aware of no increase in the severity of these symptoms; they are much as they were when they first came on.

Past history.—He has always been a healthy man, has suffered from no acute illnesses, and denies venereal disease.

Family history.—Both parents were healthy and had no sign of myotonia and no muscular wasting. Two children died as babies—one of whooping-cough, one of bronchitis. One child was stillborn. Two other members of the family survived till adult life. One of these, a sister, is the second of our cases. The other, a brother, died at the age of 47 of some wasting disease of the lower limbs, which, according to his brother, was diagnosed as "locomotor ataxia." He is said to have walked with a steppage gait and to have had especial difficulty in getting up and down stairs. He constantly complained of sharp, shooting pains in the legs, but his gait was not unsteady, even in the dark. For a great many years before his death he had the same difficulty in relaxing his grasp as that of which his brother now complains. It seems probable that he suffered from the disease which now affects his brother, and this probability is heightened by the case recorded by Rossolimo, in which the diagnosis of tabes dorsalis had been made on the strength of similar symptoms.

Present condition.—He is a thin, spare man of small frame. The pupils are equal and regular; they react well to light and on convergence. All ocular movements are of good range and there is no ptosis or nystagmus. The face is strikingly smooth and expressionless. The orbiculares palpebrarum are very weak and there is some weakness of the orbicularis oris. Attempts to keep the eyes closed against even very slight resistance are unsuccessful, and he cannot whistle or blow his cheeks out properly. The temporal regions are hollowed out, the temporal muscles being apparently diminished in bulk and contracting feebly; the masseters, however, contract well and are not wasted. The tongue and palate are normal and the vocal cords move well. Both sterno-mastoids are completely atrophied, but all the other muscles of the neck are well developed.

The muscles of the shoulders and upper arms are neither wasted nor hypertrophied and all movements at the shoulders and elbows are good in range and power. There is some wasting of the whole forearm in both limbs, especially along their ulnar borders. Flexion and extension at the wrist are feeble, but the grip is fairly good. There is no wasting of any of the small hand muscles and their power is not diminished. When the patient is asked to grasp an object he does so quickly and with fair force, but when he is told to let go there is a considerable pause before he is able fully to relax or extend the fingers. The first and second fingers extend first and these are followed, after an interval of some seconds, by the third and little fingers. The difficulty in extending the fingers is due to the slow relaxation of the flexor tendon and not to any weakness of the extensors. If the patient is now asked to repeat this movement, he does so well and relaxation is more rapidly accomplished, and by repeating the movement several times he is able to relax his grasp at once. If he simply flexes the fingers without force he is able to relax at once. The stronger the original grasp the slower the relaxation.

This slow relaxation is not well shown in any muscle, except the flexors of the fingers, but is present to a slight degree in the small muscles of the hand. The biceps, however, contracts powerfully and relaxes at once.

The muscles of the trunk and abdomen are intact. In the thighs there is almost complete atrophy of the vastus internus, and a less advanced atrophy of the vastus externus in both limbs, but in both limbs the rectus femoris has escaped atrophy. There is no wasting of any other muscles of the thighs, or of the muscles below the knees.

The range and power of all movements at the hip and ankle are good.

Flexion at the knee is powerful, but extension is greatly impaired.

The gait is normal, so far as we could observe, in spite of the before-mentioned complaint of a feeling of stiffness in the movements of the lower limbs when he first starts walking. There are no sensory changes. The knee-jerks are absent in both limbs, but the ankle-jerks are active. The plantar responses on both sides are flexor.

Electrical reaction.—The most striking feature with regard to the electric reactions is the very marked diminution of irritability of the muscles generally to the faradic current. It requires a far stronger current to produce a contraction in the muscles of the patient than in corresponding muscles of the normal individual. On first stimulating the flexor muscles of the fingers there is a brisk contraction and a relatively slow relaxation. We have been unable to time the period of relaxation, but it is probably about three to five seconds.

The interossii also showed the slow relaxation.

The leg muscles needed a stronger current than normal to produce a contraction, but relaxation was quick.

To galvanism, again, a general diminution of excitability was noticed, but no other change was present. On the face he could not bear a current strong enough to produce a contraction of the muscles.

The muscles did not give the myotonic reaction as described by Erb in Thomsen's disease.

Case 2.—A. L., aged 46, sister of the above. This patient, who has been otherwise a strong, healthy woman, states that as long as she can remember she has had a difficulty in relaxing her grasp. The condition was most pronounced when she was 20 years old; since then it has improved somewhat. She is subject, too, to a difficulty in opening her eyes the first thing in the morning, and to a stiffness of the jaws in eating. This difficulty has in great part passed off during the last few years.

Present condition.—The patient is a well-nourished, healthy woman. The pupils are equal and regular, they react briskly to light and on convergence. The ocular movements are good. There is no ptosis or nystagmus.

The masseters and temporals are not wasted, and their power is good.

The face is smooth and expressionless, and there is a great weakness of the orbiculares palpebrarum. The orbicularis oris is normal. The tongue, palate and vocal cords are unaffected.

Both sterno-mastoids are completely atrophied, but the other muscles of the neck are intact. The muscles of the shoulders, arms, forearms, hands, trunk, and lower extremities are well developed and of good power. Nowhere is there any hypertrophy. Sensation is intact.

The knee-jerks and ankle-jerks are active; the plantar reflexes give a flexor response. The gait is natural.

She has precisely the same difficulty in relaxing her grasp as her brother, but does not show myotonic symptoms in any other muscles, though she gives a definite history of difficulty in opening her eyes in the morning. We were unable to examine the electrical reactions in this case.

Case 3.—H. F., male, aged 56. This patient came under observation at the National Hospital in May, 1908, having been referred to one of us (F. E. B.) for an opinion. He had been shown at the Laryngological Section of the Royal Society of Medicine on May 1, 1908, for abductor paralysis of the right vocal cord, by Mr. Clayton Fox.

The patient gave the following history of his condition: For many years he has had a difficulty in relaxing his grasp. He cannot date the onset of this difficulty; he can only say that he has had it a great many years, but that he did not suffer from it in childhood. He has, and has had for many years, some difficulty in first starting out to walk in the morning, which soon passes off, and is not felt again during the day. He thinks the difficulty is due to an unsteadiness, rather than a stiffness in his limbs.

For some years he has had a difficulty in walking down stairs. No definite history of the date of onset of the muscular wasting can be obtained.

In December, 1907, he had an acute illness, which was diagnosed as congestion of the liver, and following immediately on this he noticed a difficulty in speaking. For this speech trouble he consulted Mr. Clayton Fox, who found an abductor palsy of the right vocal cord, and referred him to Dr. Batten for an opinion.

Past history.—He had a cellulitis of the forearm thirty years ago and erysipelas of the face ten years ago. He denies venereal disease, has always been a non-smoker, and for the last forty years a total abstainer. He has been married many years, but has no children.

Family history.—His father, who had no muscular wasting, or signs of myotonia, died at the age of 64 of a tumour of the gut. His mother had throughout her life a difficulty in relaxing her grasp, which improved as she grew old: she had no muscular wasting and died at the age of 82 of "senile decay." He has had two brothers and two sisters. Both sisters are dead, one as an infant, one in adult life of some unknown cause. One brother died at the age of 52 from bronchitis, one is still alive and in good health. None of his brothers or sisters had myotonic symptoms or muscular atrophy. He knows nothing about any other relatives.

Present condition.—The patient is a thin, old-looking man with a myopathic facies. The pupils are equal and regular, and react well to light and on accommodation. All ocular movements are good in range, and there is no ptosis or nystagmus. The face is smooth and expressionless. There is great weakness of the orbiculares palpebrarum, and some weakness of the orbicularis oris. The temporal muscles can be felt to contract, but their bulk is apparently diminished. The masseters contract forcibly, and are not wasted. The tongue is protruded straight without tremor, and is not wasted. The palate moves well and equally on the two sides. There is a partial abductor palsy of the right vocal cord.

Both sterno-mastoids are completely atrophied, and there is some wasting of the upper parts of both trapezii. All movements of the head and neck are performed with fair power.

There is no wasting or hypertrophy of any muscle about the shoulders and upper arms, and all movements about the shoulders and elbows are free and of good power. Both forearms are flabby and slightly wasted, the wasting affecting the flexors and extensors to an equal degree. All movements about the wrist are of fair power. There is no atrophy of any of the small hand muscles. When the patient is asked to grasp an object, he does so well and powerfully, but when asked to relax his grasp, he does so almost at once with the first and second fingers, but the third and fourth fingers relax slowly. The condition is exactly similar to that seen in the last patient, but is less definite.

There is no obvious wasting of any of the trunk muscles, but there is a slight degree of lordosis in the lumbar and lower dorsal regions.

The thighs are well developed, except for an almost complete atrophy of the vastus internus and externus in both limbs. The rectus femoris is intact, and no other muscles of the thighs are wasted. All movements of the thigh are powerful. Flexion at the knee is powerful; but extension is very feeble. The calf muscles are flabby and large, out of proportion to the development of the general musculature of the body. The same condition of hypertrophy is observed in the anterior tibial groups in both limbs.

All movements about the ankle are of good range, but the power of all movements, especially dorsiflexion of the feet, is very poor. This loss of power is emphasized by contrast with the large bulk of the muscles.

The knee-jerk is abolished on the left side, it is present but very difficult to elicit on the right: the ankle-jerks are absent on both sides. The plantar responses are flexor.

There are no sensory changes. In walking he lifts the feet unduly high, and flings the legs far forwards. He rises from the lying posture in the manner so constantly seen in pseudohypertrophic paralysis.

Electrical reaction.—The most striking feature with regard to the electrical reaction is the definite diminution of irritability of the muscles to faradic stimulation. This is present not only in the muscles of the arms, but also in those of the legs.

The only muscle which tends to give a slow relaxation is the flexor of the fingers, and the reaction is of the type Pässler described as a modification of the myotonic reaction, *i.e.*, both slow contraction and slow relaxation.

Case 4.—F. J., male, aged 44. This patient was admitted to the National Hospital in October 1908, under the care of Dr. Risien Russell, to whom we are indebted for permission to record the case.

Two years before admission he felt, while walking, a sudden weakness in the right knee, and from that time he noticed an increasing weakness about that knee. In August, 1907, the left knee became similarly affected, and on one occasion he fell when tired. For many months he has been unable to run at all, because of this tendency for his knees to give way under him. He has an especial difficulty in coming down stairs, which he attributes to a loss of power in the muscles about the inner side of the thighs. He has noticed no weakness about the arms or trunk, but the forearms have been wasting for years, though they seem quite strong. He has had during the last few years a difficulty in rising from the stooping posture without the help of his arms.

No history of the date of onset of the myotonic symptoms to be described could be obtained.

Past history.—He had rheumatic fever when 11 years old; this illness left his heart weak. He had pneumonia in 1892.

Nine years ago he fell unconscious on two occasions. These unconscious attacks were attributed to heart trouble, and since then he had been unable to do any hard manual labour. He denies venereal disease. He has been married eighteen years and has two healthy boys. His only other child, a girl, died at the age of 11. She was "mentally affected."

Family.—We were unable to obtain any information as regards the existence of any similar condition in any relatives.

Present condition.—He is a thin, rather wasted man. The pupils are equal and regular, and react to light and on convergence. All ocular movements are good in range. Extreme lateral movements of the eyeballs bring out occasional slow nystagmoid jerkings. The masseters are greatly wasted and contract feebly; the temples are hollowed and the temporal muscles can hardly be felt. The face is expressionless; the eyes can be closed completely, but with poor power. The lower facial muscles move well on both voluntary and emotional efforts.

The sterno-mastoids are completely atrophied on both sides; all the other muscles of the neck are well developed and powerful. Lateral and forward movements of the head are feebly performed, though the range of movement is good.

The shoulder and upper arm muscles show nowhere either atrophy or hypertrophy, and all movements at the shoulders and elbows are powerful. The muscles of the forearms, especially the extensors, are wasted. Flexion and extension at the wrist are poor in power, and the grip is feeble on both sides. The difficulty in relaxing the grasp after making a fist is precisely similar to that seen in the first case. The intrinsic hand muscles are not atrophied, and all movements of the thumbs and fingers are strongly performed.

The muscles of the trunk are unaffected. The muscles of the lower limbs are well developed, except for a complete atrophy of the vastus internus and externus in both limbs, the rectus femoris remaining intact. The muscles below the knees are well developed. All movements about the hips are of good power. Flexion at the knees is powerful in both limbs, but extension is very feeble. Dorsiflexion at the ankle is relatively not so powerful as other movements about the joint. Sensation is unimpaired.

The knee-jerk is absent in the right limb; in the left it can be obtained on reinforcement, but does not overcome the inertia of the joint. Both ankle-jerks are active, and the plantar reflexes are flexor. The gait is normal.

Electrical examination of the muscles was not made in this case.

Case 5.—F. W., male, aged 24. This patient was in the National Hospital in 1906, under the care of Dr. Tooth. Since then he has been under the care of Dr. Farquhar Buzzard as an out-patient at the Royal Free Hospital, and was shown by him at the Neurological Section of the Royal Society of Medicine as "an unusual case of muscular atrophy." To both we are indebted for the use of the clinical notes.

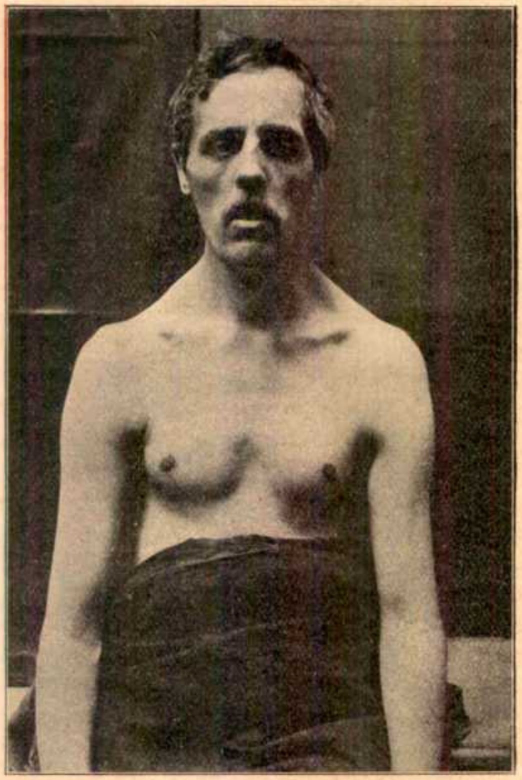


FIG. 1.

CASE 5.—Showing the atrophy of the sterno-mastoids and the myopathic facies.

The patient had always enjoyed good health till the onset of his present illness. He denies venereal disease, but admits having been a heavy drinker. There is no history of muscular wasting or of myotonic symptoms in any relatives. In October, 1905, he had a severe attack of rheumatism, involving the knees, ankles, and shoulders; and when convalescent from this illness he found that he could not walk without stepping very high. This difficulty in walking has become progressively worse. It was not until 1907 that he began to have any difficulty in relaxing his grasp.

Present condition.—The patient is a tall, spare man. The pupils are equal and regular, and react to light and on accommodation. All ocular movements are good, and there is no ptosis or nystagmus. The masseters and temporals are strong, and not atrophied. There is great weakness of the orbicularis palpebrarum, and less marked weakness of the orbicularis oris. The tongue, palate and vocal cords are normal.

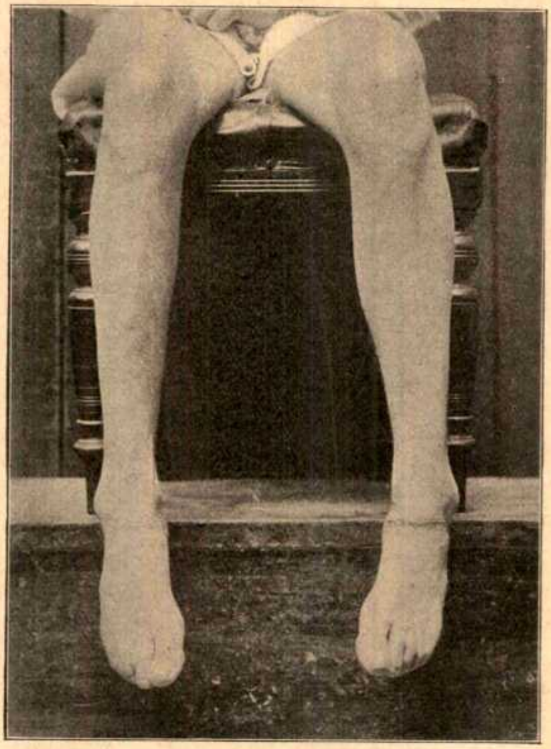


FIG. 2.

CASE 5.—Showing the moderate degree of wasting of the peronei and anterior tibial groups.

The sterno-mastoids are almost completely atrophied, but all other muscles of the neck are well developed (fig. 1). The muscles of the shoulders and upper arms are well developed and of good power (with the exception of the supraspinati, which are considerably wasted). Both forearms are thin and wasted along their inner borders. Flexion and extension at the wrist (especially extension) are feeble and the grip in both hands is poor. The small hand muscles show no atrophy.

The muscles of the trunk are intact, as are also the muscles of the thigh, with the exception of the vastus internus, which is wasted in both limbs. The anterior tibial group are wasted, but not to an extreme degree (fig. 2). All movements of the hip and knee are of poor power. At the ankles neither dorsiflexion nor eversion can be performed. Extension is fair in power. There are no sensory changes.

The knee-jerks are present, the ankle-jerks absent. The myotonia is shown only in a difficulty in relaxing the grasp, and is precisely similar to that we have already described in Cases 1 and 4.

The muscles of the shoulder and upper arm react briskly to faradism and galvanism. The flexors of the wrist and fingers react well to faradism, but show in a marked degree the myotonic reaction. The extensors of the wrist react very poorly both to faradism and galvanism, but show no myotonic phenomena. The small hand muscles react poorly both to faradism and galvanism, and to the former give a modified myotonic reaction, *i.e.*, both slow contracture and slow relaxation. The extensors of the foot react neither to faradism nor galvanism.

Summary of Clinical Symptoms.

All these cases resemble one another closely, not only in the presence of myotonic symptoms, but also in the distribution of the atrophy. Affection of the facial musculature and atrophy of the sterno-mastoids is common to all of them. In the second case these muscles are the only ones involved, but the presence of the affection in a more advanced degree in a brother leaves no doubt as to the nature of the condition. In the remaining four cases the muscles of the shoulders and upper arms and the intrinsic muscles of the hands escape, but the forearms are affected by a general wasting, and the weakness is not confined to any particular group of muscles.

The muscles of the trunk are unaffected, but in all atrophy is present in the lower limbs. In Cases 1 and 4 the distribution of this wasting is identical—the muscles below the knee have escaped entirely, as have also the muscles of the thigh, with the exception of the vastus internus and externus, both of which—especially the vastus internus—are definitely atrophic. Case 3 shows a similar atrophy of the vasti, but there is in addition a considerable weakness of all the muscles below the knees, especially of the dorsiflexors of the feet, associated, not with wasting, but with an apparent hypertrophy of the muscles. In Case 5 the thigh muscles have escaped atrophy, but here there is wasting and weakness of all muscles below the knee, especially of the dorsiflexors of the feet, which are completely paretic.

Case 3 cannot unreasonably be regarded as forming a link between Cases 1 and 4 on the one hand and Case 5 on the other, and the history which F. W. gives in the case of his dead brother suggests that in this instance also weakness of the dorsiflexors of the feet was a prominent feature.

The unusual and apparently irregular distribution of the wasting in cases of myotonia atrophica has been commented on by most observers. Hoffmann pointed out the almost constant involvement of the face, the frequent affection of the masseters and the escape of the intrinsic muscles of the hands, but he does not appear to have laid any considerable emphasis on this distribution, for he makes irregularity of distribution one of the points of his argument in favour of the condition being primarily one of myotonia. The cases which we have here recorded seem, however, so strongly to suggest that a characteristic distribution may exist in the disease, that we have investigated all the cases which have been recorded with a view to determining how closely they correspond to our own.

We have succeeded in discovering twenty-nine recorded cases. Three presented symptoms which suggested strongly some complicating lesion of the nervous system. Frohmann's case showed nystagmus and Rombergism, Lortat-Jacob and Thaon's an extensive anæsthesia, and Pelz's nystagmus and hyperalgesia of the nerve-trunks. Of the remaining twenty-seven cases, fifteen present a distribution approximating closely to that which we have here described. These fifteen cases, with our own five, seem to form so definite a group that we give a short abstract of their clinical features in an appendix. In the remaining twelve cases the atrophy differed so widely in distribution from our own that we have neglected them, for it is our object not so much to discuss the whole subject of myotonia atrophica, as to suggest the possibility of there existing a form of myopathy differentiated clinically from the classical types, as much by the distribution of the wasting as by the presence of the myotonia.

From an analysis of our own cases and those in the literature which most closely resemble the "type" described we are able to draw the following conclusions with regard to the leading features of the condition.

(1) *Familial Characters.*

In several of these cases the disease occurs in more than one member of a family. Hoffmann [8] recorded the disease in a brother and sister, Pässler in two brothers, Ramsay Hunt in two brothers, and Batten and Gibb a family in which a brother and sister were certainly affected and probably a second brother who did not come under observation. Among the cases which we have here collected there was no instance of the presence of myotonia without atrophy in members of the same generation.

Direct heredity could not be traced in any case, but Rossolimo [17] records the case of a man whose father had (according to the son) presented symptoms of myotonia; and one of our patients states definitely that his mother suffered throughout her life from a difficulty in relaxing her grasp. In both these parents there was, so far as is known, no muscular wasting. In no instance was a history of myopathy in ascendants or the collaterals of ascendants obtained.

(2) *Etiology.*

The disease arises in most instances without apparent cause. In two cases reported by Hoffmann and Fürnrohr the condition was attributed to exposure to severe cold. In Rossolimo's case the condition followed an acute illness characterized by a painful swelling of the knee-joints, and in one of our own an attack of acute rheumatism closely preceded the first onset of the weakness.

Such apparent causes of origin, if they have any significance at all, can scarcely be regarded as other than secondary in determining the onset of the disease.

(3) *Sex.*

The disease is much commoner among males. Few as the recorded cases are, they can leave no doubt on this point. In only three out of twenty cases were the patients women—viz., in Hoffmann's, Batten and Gibb's [2], and Kleist's [10] cases—and it is interesting that in two of these the symptoms were slight and had escaped observation till the presence of the affection in a more marked degree in other members of the family had directed attention to the condition.

(4) *Age of Onset.*

The symptoms make their appearance in early adult life; in the majority of cases between the ages of 20 and 30. The earliest age at which symptoms have appeared is 15 (Kleist [10]). In a few instances the onset has been delayed till beyond the thirtieth year. The myotonia usually precedes the atrophy, but not invariably. In the two cases recorded by Ramsay Hunt, muscular wasting had been noticed for many years before myotonic symptoms showed themselves, and in one of our own the myotonia did not appear till 1907, though the wasting had reached an advanced degree in 1906.

(5) *Distribution of the Atrophy.*

Atrophy of the facial musculature is almost constant (sixteen in twenty). In all our cases the features had the expressionless immobile character seen in the Landouzy-Dejerine type of myopathy, and the weakness of the facial muscles, especially of the orbicularis palpebrarum and the orbicularis oris, was manifest on all attempts at voluntary movement.

In half the cases (ten in twenty) the masseters and temporals were also affected. In one instance (Nonne [13]) the atrophy had reached so high a degree as to have caused a bilateral dislocation of the jaw.

The sterno-mastoids were also frequently affected (thirteen in twenty). In these thirteen cases the sterno-mastoids on both sides were atrophic; this atrophy contrasts forcibly with the almost constant escape of the trapezii, which were atrophic in three instances only. The other muscles of the neck have always escaped. The muscles of the shoulder and upper arm were rarely involved. In some few cases there has been noticed a wasting of the supraspinati and infraspinati; the deltoid was atrophied in a slight degree in one instance only; the other muscles about the shoulders have always escaped. The muscles of the upper arm were affected in four of these cases, but only to a slight degree. The biceps generally suffered most where the upper arm was affected. But the most constant feature in all these cases is the involvement of the forearm musculature. The atrophy did not involve any particular group of muscles, both flexors and extensors suffered, though, as a rule, the flexors were weaker and more wasted than the extensors.

In striking contrast to the wasted condition of the forearms is the non-atrophic state of the intrinsic muscles of the hand, which in most cases were preserved. In only four cases was there any atrophy of these muscles. It involved especially the thenar and hypothenar eminences.

The trunk muscles in all instances escaped, but the legs were almost invariably affected; of the thigh muscles, the extensors of the knee far the most commonly (nine in twenty). The adductors of the thigh were involved in one instance only, and the other muscles of the thigh invariably escaped. Where the atrophy invaded the quadriceps extensor femoris it showed a tendency to affect the vastus internus and externus and leave the rectus femoris intact. This is strikingly shown in three of our cases.

But of the lower limb muscles, those below the knee were the most commonly affected, and of these the peronei and dorsiflexors of the foot more commonly than the calf muscles. Weakness of the dorsiflexors was present in ten of these nineteen cases, and in one instance (Lannois [11]) the forearms and dorsiflexors of the foot were alone involved. In nine out of twenty cases the calf muscles were weak; but as a rule the weakness was less marked than in the dorsiflexors and in no case were the calf muscles affected without involvement of the dorsiflexors.

In three instances the laryngeal muscles were affected. In Nonne's case there was " a paresis of the vocal cords " ; in Siemerling's paralysis of the adductors (thyro-arytænoideus), and in one of our own the patient first sought advice for an abductor palsy of one of the cords.

(6) *The Myotonia.*

The myotonic phenomena are shown far more commonly in a difficulty in relaxing the grasp than in any other voluntary movement. In all cases this phenomenon has been present and in most instances has been the only voluntary movement showing the presence of myotonia. Even in cases in which the myotonia is not confined to this particular movement, it is far more conspicuous in it than in any other. Rossolimo's case, in which this disability involved to a considerable degree all movements of the upper arm and neck, is the only one recorded in which the myotonia was at all conspicuous in other parts of the body. In all other instances the myotonic phenomena were slight and most frequently apparent in the muscles supplied by the cranial nerve, the facial muscles, the muscles of mastication and the tongue. Myotonic phenomena are not common in lower limbs, and when present are always slight in degree.

Further, the myotonic appears to bear no relation to the atrophy, so far as voluntary movements are concerned. Even in instances in which the atrophy in the lower limbs is considerable, myotonic phenomena are generally absent.

APPENDIX.

Nonne [13].

Male, aged 40. Atrophy of facial muscles, masseters, neck (muscles not specified), shoulder-girdle, forearms, thighs and peronei. Paresis of vocal cords.

Myotonia in limbs and eyelids. Mechanical and electrical myotonia reaction in all muscles not atrophied and in some atrophic muscles.

Knee-jerks and ankle-jerks absent, and deep reflexes in arms diminished.

Pässler [15]. Two brothers.

Case 1.—Aged 35. Myotonia first noticed at puberty. Weakness and atrophy of hands some years later.

Atrophy of facial muscles, masseters, temporals, sterno-mastoids, supraspinati and infraspinati, flexors and extensors of forearms, intrinsic muscles of the thumb and of fourth interossei.

The legs are said to have been slightly weak, but there is no record of atrophy in the lower limbs.

Pronounced myotonia in hands and arms.

Case 2.—Aged 33. Myotonia first at 18. For the last few years wasting of hands.

Atrophy of facial muscles, masseters, temporals, sterno-mastoids, supraspinati, infraspinati, and of interossei muscles of the feet.

Hoffmann [8]. Brother and sister.

Case 1.—Male, aged 26. Has three living sisters; two of them seen by Hoffmann: one of these healthy, the other is the second case recorded.

Weakness of grip noticed at the age of 20, attributed to exposure to cold. Steadily increasing weakness and wasting of forearms. The weakness of the face probably present since the age of 18. Difficulty in speaking present for a year; no record of date of onset of myotonia in hands, but said to have been healthy as a child.

Atrophy of facial muscles, masseters, sterno-mastoids, flexors of left arm, muscles of forearm in both limbs more advanced in the flexors than the extensors. The lower limbs are unaffected.

Myotonia present in forearm and hand muscles, slighter in degree in tongue and upper arms. Deep and superficial reflexes all present. Myotonic, electrical and mechanical reactions in the muscles of the upper extremities, but not in those of the lower extremities.

Case 2.—Female (sister of above), aged 30. Considered herself healthy.

Atrophy of facial muscles, masseters, sterno-mastoids, and the muscles of the forearms, especially the flexors.

The lower limbs involved.

The hands show a typical myotonia, and the myotonic electrical reaction is present in the forearm muscles. Deep and superficial reflexes present.

Fürnrohr [6].

Case 1.—Male, aged 33. No history of a similar condition in family. When 20 years old noticed a difficulty in relaxing his grasp; twelve years later began to lose power in his legs.

Atrophy of the forearm and thigh muscles, the lower leg being well developed. The knee-jerks are absent. Myotonia in forearm muscles.

Case 2.—Male, aged 50. For some years slowly progressive muscular atrophy. Atrophy involving the facial muscles, flexors of the head, shoulder muscles, forearms and hands. Also weakness and atrophy of the thighs and legs. He complained of no myotonic symptoms, and it was only after careful examination that these were detected.

Rossolimo [17].

Male, aged 37. No history of similar condition in parents or other relatives. Always healthy till onset of present condition.

When 22 years old he began to show a progressive wasting and feebleness of the whole body; ten years later the right knee, without apparent cause, became red, swollen, and painful. The other joints were unaffected. Two months after this he noticed a progressive weakness about this knee, and shortly afterwards the left knee began to get weak too, and he frequently tripped and fell. He noticed, too, a stiffness about the knees when he first started walking, which passed off after a few steps. At the same time as the difficulty in walking commenced he began to have a stiffness about all movements of the upper limbs, which passed off after repeated movements. The power of the movements of the upper limbs began to fail at the same time.

Present condition.—Atrophy involving face masseters, forearms, extensors and adductors of thighs with symmetrical wasting of muscles below the knees.

The myotonia is shown principally in the flexors of the forearm, but is present in movements of the upper arm, neck, and lower limbs..

The knee-jerks are difficult to elicit, and the ankle-jerks are abolished.

Lannois [11].

Male, aged 37. No history of a similar condition in any relatives.

When 3 years old it was observed that he threw out his feet in walking. Two years later lightning pains in lower limbs; first myotonia noticed in movements of hand, three years after difficulty in walking began.

Present condition.—Atrophy. Forearms, not localized to any special group of muscles, dorsiflexors of feet, and to a less degree of calf muscles. Myotonia shown chiefly in forearm muscles, but to a lesser degree in movements of lower limbs, and in talking and chewing.

The deep reflexes are absent.

Noguès and Sirol [14].

Male, aged 33. His father had some difficulty in starting to chew. No other relatives affected.

When 17 years old noticed a difficulty to chew, and also a difficulty in relaxing his grasp. At the age of 28 he began to tire easily in walking, and noticed a stiffness about his legs on first starting to walk.

Present condition.—Slight general wasting and weakness of arms. Atrophy of inner belly of gastrocnemius in both legs and of the tibialis anticus and common extensor of the toes, and to a less degree of the peronei. Very obvious wasting of the vastus internus in both limbs.

The deep reflexes are diminished, but not abolished.

Schönborn.

Male, aged 22 (from Hoffmann). Always slower than other men, and his speech has always been nasal. For the last two years has had progressive weakness and wasting of muscles of hands and forearms.

Present condition.—Atrophy of hand and forearm muscles, and of the extensors of the lower limbs.

Siemerling [18].

Male, aged 31. When 26 years old wasting of forearms began. When 27 began to suffer from a feeling of stiffness in muscles as soon as he wished to perform a movement, passing off after repeated efforts.

Present condition.—Atrophy affecting face masseters, tongue, sterno-mastoids, trapezii, especially middle and lower part, supraspinati and infraspinati, deltoids, especially middle portion upper and lower arm. Thighs powerful, lower legs weak.

There is also weakness of the adductors of the vocal cords.

Knee-jerk absent in left leg; obtained only with reinforcement in the right one.

Ramsay Hunt [7].

Two brothers. Another brother, now dead, is said to have presented similar symptoms.

Case 1.—Aged 44. For ten years has had weakness of the hands, and for six years of the lower extremities. Three years ago myotonia phenomena first noticed.

Present condition.—Atrophy involving face, upper arm, forearms and muscles below the knees, especially the dorsiflexors. Myotonic phenomena in forearm muscles, to a less degree in the movements of the lower limbs.

The deep reflexors are abolished.

Case 2.—Aged 41. When 23 years old noticed weakness of the hands and arms. Not till the age of 33 did myotonic phenomena show themselves. Then they appeared in the movements of the hand, and about the same time stiffness and weakness of the lower limbs appeared.

Present condition.—Atrophy involving face, upper arms, forearms, and muscles below the knee, especially the dorsiflexors.

Myotonic phenomena only in movements of hands.

Deep reflexes greatly diminished.

Elzist [10] has described a case under the title, "Nachdauernde Muskelkontraktionen" of a woman aged 29.

She was well till 7 years old, when she had gastric fever, followed by chorea, and when about 15 years old she had some unsteadiness of the right hand.

About this time she noticed that the hand "stood still," and that when she grasped an object she could not let go. The patient continued at work till she was 26. The increasing weakness made her give up her work.

On examination, a lingering contraction of the flexor muscles of the hand was observed. The more powerful the contraction the longer is the period of relaxation, but repetition of the movement does not diminish the tendency to this lingering contraction. The same phenomenon is found in some other muscles of the body.

The muscles are readily exhausted on voluntary movement, but there is no myasthenic reaction to electrical stimulation.

There is atrophy in the dorsal forearm muscles and the dorsiflexors of the feet, of the sterno-mastoids and of the facial muscles. Scoliosis, pes equinus, and claw-foot were also present.

There was slight sensory disturbance on the outside of the thighs.

The author argues at some length that the myotonic condition of the muscles is due to affection of the fronto-pontine-cerebellar tract.

The case is one which does not correspond in a good many points with the cases we have described. It is especially noticeable that the lingering contraction of the muscles is not removed in this case by repeated movements, as it was in other cases.

REFERENCES.

[1] Batten. "Case of Myopathy, exhibiting Slow Relaxation of Muscles after Muscular Action," *Brain*, 1906, vol. xxix, p. 414.

[2] Batten and Gibb. "Two Cases of Myotonia Atrophica, showing a Peculiar Distribution of Muscular Atrophy," *Proc. Roy. Med. Soc.*, 1908, Neurological Section, p. 32.

[3] Buzzard. "An Unusual Case of Muscular Atrophy," *Proc. Roy. Soc. Med.*, 1908, Neurological Section, p. 33.

[4] Fox. "A Case of Laryngeal Paralysis," *Proc. Roy. Soc. Med.*, Laryngological Section, 1908, p. 107.

[5] Frohmann. "Ueber Thomsensche Krankheit mit Muskelatrophie," *Deut. med. Woch.*, 1900, V.B., S. 6.

[6] Furnrohr. "Myotonia Atrophica," *Deut. Zeit. f. Nervenh.*, 1907, xxxiii, S. 25.

[7] Hunt. "Myotonia Atrophica," *Journ. Nerv. and Ment. Dis.*, 1908, xxxv, p. 269.

[8] Hoffmann. "Ueber Myotonie," *Neurol. Centralb.*, 1905, S. 576.

[9] *Idem.* "Zur Lehre von der Thomsenschen Krankheit mit besonderer Beruchsichtigung des dabie vorkommenden Muskelschwundes," *Deut. Zeit. f. Nervenh.*, 1900, xviii, S. 198.

[10] Kleist. "Ueber Nachdauernde Muskelkontraktionen," *Journ. f. Psych. u. Neurol.*, 1907, x., S. 95.

[11] Lannois. "Myotonie avec atrophie musculaire," *Nouv. Iconogr. de la Salp.*, 1904, p. 450.

[12] Lortat-Jacob et Thaon. "Atrophie musculaire avec réactions electriques de la maladie de Thomsen," *Arch. de Neurol.*, 1905, xx, p. 132.

[13] Nonne. "Combination von Myotonie und Dystrophia Muscularis," *Neurol. Centralb.*, 1905, S. 142.

[14] Noguès et Sirol. "Maladie de Thomsen à forme fruste avec atrophie musculaire," *Nouv. Iconogr. de la Salp.*, 1899, xii, p. 15.
[15] Fassler. "Zwei Bruder mit Amyotrophischer Myotonie," *Neurol. Centralb.*, 1906, S. 1064.
[6] Pelz. "Ueber Atypische Formen der Thomsenchen Krankheit," *Arch. f. Psych. u. Nervenkrank.*, 1907, xlii, S. 704.
[7] Rossolimo. "De la myotonie atrophique," *Nouv. Iconogr. de la Salp.*, 1902, p. 63.
[8] Siemerling. "Fall von Myotonia congenita, Muskelatrophie und Myasthenia," *Munch. med. Woch* , 1905, lii., S. 1072.

NOTE ON THE MOVEMENTS OF THE TONGUE FROM STIMULATION OF THE TWELFTH NUCLEUS, ROOT, AND NERVE.

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DURING the winter of 1907-8 it was my privilege to be associated with Sir V. Horsley in an investigation into the functions of the inferior olive.

To reach this important group of cells it was necessary for the stimulating needle to pass through the fibres of the twelfth root, and we frequently observed that while stimulation of some of these fibres caused the tongue to protrude to the right, stimulation of other fibres of the same root produced a strong protrusion of the tongue to the left.

In the course of my investigations, which have been continued at McGill during the past year, I have always been puzzled by this protrusion of the tongue to the *left* on stimulating the left twelfth root, for it is generally understood when the tongue is protruded from the left side of the mouth, or into the left cheek, that this movement is due principally to the protruding action of the right half of the tongue assisted by the contraction of the left half [1].

The purpose of this paper is to show that the protrusion of the tongue from the left side of the mouth, and the protrusion of the tongue into the left cheek, may be accomplished by the muscles of the left half of the tongue alone without any assistance from the right half.

In these experiments Dr. Clarke's stereotaxic instrument [3] has been employed. The animal was deeply anæsthetized and the instrument adjusted. The medulla was exposed from the foramen magnum to a few millimetres below the superior border of the atlas so as to have free access for the needle to the twelfth nucleus and root. The movements of the tongue were observed on stimulating (1) the twelfth nucleus, (2) the root in its course through the medulla, and (3) the twelfth nerve in the neck.

On stimulating the left twelfth nucleus (*mutatis mutandis* the same is true of stimulation of the right nucleus), the following movements were observed :—

The tongue was protruded to the opposite side (right); the tongue was curled upwards and over to the right canine tooth. But the most unexpected reaction was a strong protrusion of the tongue from the mouth on the left side.

When the left twelfth root in the medulla was stimulated, similar movements were produced. The tongue was curled upwards and to the right or opposite canine tooth, while stimulation of other fibres caused the tongue to protrude from the angle of the mouth *on the same (left) side.*

The most frequent reaction on stimulating the (left) twelfth nerve in the neck was *a strong protrusion of the tongue out of the mouth on the same side.* Other reactions were: protrusion with the tip of the tongue strongly to the right or opposite side, and curling up of the tip, now to the right and at other times to the left.

EFFECTS AFTER MEDIAN SAGITTAL SECTION OF THE TONGUE.

To exclude any assistance from the right half of the tongue in these movements, the tongue was then divided into two halves along the raphé and the same parts were again stimulated in the nucleus, root, and nerve.

On *stimulating the left nucleus*, the *left half* was protruded to the *right*; the left half could curl up and over to the right canine with no movement of the right half. On reaching the spot that caused on stimulation a strong protrusion of the tongue to the left, *i.e.*, to the same side, it was interesting to see how this movement was produced, for the left half was retracted in a straight line and the right half was strongly protruded—showing the close association of the two nuclei.

This conjugate action of the two separated halves of the tongue was first described by Beevor and Horsley [2] on stimulating the upper part of the tongue area in the right hemisphere.

Stimulation of the left twelfth root caused the left half to curl up to the opposite (right) canine; stimulation of other fibres produced a strong protrusion of the left half to the right; and on stimulation of this left root in another area, the left half was protruded from the left angle of the mouth while the right half remained motionless.

When the *nerve* was stimulated the movements of the left half of the tongue were the same as those observed before the tongue was divided, but now were performed by the left half only.

It will be noticed that when the tongue was protruded from the left side on stimulating the *left nucleus*, that this movement was due to the retraction of the left half and the protrusion of the right. But when the *root* or the *nerve* was stimulated the protrusion from the left side was accomplished by the muscles of the left half alone.

Having demonstrated the actions of the tongue in the cat, and feeling that a carnivorous animal was probably not suitable for comparison with man, I concluded this investigation by repeating these experiments on a bonnet monkey.

The twelfth nucleus, root, and nerve were stimulated, first with the tongue in a normal state, and then after dividing it into two halves.

All the movements corresponded with those found in the cat and may be summarized as follows :—

(1) The half of the tongue that is stimulated can be protruded to the opposite side as far as the canine teeth.

(2) Can be protruded straight.

(3) Can be protruded from the same side.

(4) Can be protruded into the cheek of the same side.

(5) The tip of the tongue can touch the teeth and gums of the upper and lower jaw on the same side, and as far as the canine teeth, upper and lower, on the opposite side.

(6) The tip of the tongue can curl upwards and backwards to the roof of the mouth on the same side.

In fact, it was clearly demonstrated that the only movements that the stimulated half of the tongue could *not* perform were : protrusion from the opposite angle of the mouth, protrusion into the opposite cheek, as well as the inability to touch the molar teeth and the gums on the opposite side.

Therefore, these experiments show that when one half of the tongue is paralysed from affection of nucleus, root, or nerve, the following symptoms are observed :—

Inability to touch the teeth, the gums, or to protrude the tongue into the cheek of the paralysed side, while there is perfect freedom of movement to the sound side, the tongue being easily protruded into the cheek of this side and the teeth and gums being readily touched ; in advanced cases secondary atrophic changes in the paralysed half may, however, greatly interfere with the movements of the sound half.

REFERENCES.

[1] BEEVOR. *Brain*, 1906, p. 487.
[2] BEEVOR and HORSLEY. *Phil Trans. Royal Society*, 1894, vol. 185, p. 56.
[3] HORSLEY and CLARKE. *Brain*, 1908, vol. xxxi., p. 1.

THE OCCURRENCE OF REMISSIONS AND RECOVERY IN TUBERCULOUS MENINGITIS: A CRITICAL REVIEW.[1]

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I. INTRODUCTION.

THE majority of the medical profession have found the termination of tuberculous meningitis so uniformly fatal that the greatest scepticism has been shown with regard to the diagnosis of cases in which there were prolonged remissions of symptoms or in which recovery occurred.

Taylor [55], for instance, writes: "Undoubtedly tubercular meningitis is in nearly all cases fatal; and the prognosis may be regarded as unfavourable in proportion to the certainty of the case being tubercular." According to Gowers [25], "doubt always hangs over the exact nature of the cases that recover, but the probable evidence as to the tubercular character of some of these cases is very strong." And in the opinion of Barlow [3], "the study of the morbid anatomy of the disease gives us the only reasonable basis on which to discuss the subject of prognosis. If the tuberculous deposition be limited to a part of the surface of the brain, there is a possibility of recovery; but if the disease becomes generalized, so that besides the invasion of the pia mater there is an extensive meningo-encephalitis with or without hydrocephalus, the chance of recovery is practically *nil*." Within the last ten years, however, methods have been more generally employed by means of which the diagnosis of tuberculous meningitis can be confirmed during life, and it is now possible to prove beyond doubt that a patient is suffering from tuberculous meningitis while he is yet alive.

The object of this paper is to sift the evidence obtainable and to show that tuberculous meningitis does sometimes recover, or that long remissions may occur during its course.

It is instructive, in the first place, to examine the statistics of institutions in which large numbers of cases of tuberculous meningitis are observed. I have therefore analysed the reports of several London hospitals, and the reports of the Vienna hospitals[1] since the year 1897. In the clinical reports of seven London hospitals issued since that year 797 cases of tuberculous meningitis are referred to, and of these 16, or approximately 2 per cent., recovered; while in the Vienna hospitals during the same period there were 1,369 cases, but of these only six, or 0·44 per cent., are reported as recoveries. Thus, of 2,166 cases diagnosed as tuberculous meningitis twenty-two only, or about 1 per cent., were discharged from hospital cured or relieved.

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[1] This paper was part of a thesis presented for the degree of M.D. to the University of Cambridge.

These figures cannot, however, be accepted without criticism, and there is no doubt that the percentage of recoveries shown in the figures taken from the records of the London hospitals is too high. I have personally examined the notes of thirteen out of the sixteen cases recorded as recoveries in the seven hospitals from the reports of which these figures were obtained, and have found that one case had been recorded as a recovery through a clerical error; two cases had been in hospital for so short a period—two days and fourteen days respectively—that they cannot be accepted as cases of tuberculous meningitis; two other cases were removed from hospital while acute symptoms were still present, so that they cannot be considered recoveries; and two were discharged unimproved. The notes in one case were missing. In four of the remaining five cases examined, the diagnosis was made on clinical evidence only; in the other there was, in addition to clinical evidence, an increase in the cell elements in the cerebrospinal fluid (polymorphonuclear cells being in excess of lymphocytes), while on two occasions the fluid proved sterile after incubation for three days; this excludes meningococcal and suppurative meningitis.

As a result of this investigation the number of *possible* recoveries reported in the statistics of the seven London hospitals given above *must* be reduced to nine.

I was, unhappily, unable to trace the records of four of these cases; in four of the remaining five cases the diagnosis was made on clinical grounds alone, and consequently they cannot be accepted for the purpose of statistics. The remaining case, in which the clinical diagnosis was supported by the existence of an increase in cell elements in the cerebrospinal fluid that remained sterile on incubation, is the only one in which there was a strong probability of tuberculous meningitis.

In any research on the subject it is essential that the nature of all the cases in which a prolonged remission or recovery is recorded should be placed beyond doubt.

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[1] *Jahrb. der Wiener Krankenanstalten.*

The diagnosis of this disease by clinical methods alone often presents the greatest difficulty, as there is no pathognomonic symptom. It is increased by the fact that the initial symptoms of infectious diseases, especially of enteric fever, pneumonia, and broncho-pneumonia, in children often resemble those of tuberculous meningitis, and in young children teething and gastric disturbances give rise to somewhat similar symptoms. Finally, other forms of infective meningitis and the meningeal symptoms in uræmia, lead poisoning, and hysterical pseudomeningitis may also lead to confusion.

Hence it is impossible to accept a diagnosis of tuberculous meningitis as certain if it is based solely on the clinical symptoms, though the probability may be very great.

II. METHODS OF CONFIRMING THE DIAGNOSIS.

To establish the diagnosis of tuberculous meningitis further steps are necessary; of these the chief is lumbar puncture. By this means we are able to obtain information as to—

(1) The tension of the cerebrospinal fluid.

(2) The appearance and chemical nature of the fluid.

(3) The presence or absence of tubercle bacilli or other organisms in the fluid by microscopical examination.

(4) The presence of tubercle bacilli in the fluid by the experimental production of tuberculosis by inoculation of a susceptible animal like the guinea-pig.

(5) The presence of tubercle bacilli by their cultivation in special media.

(6) The presence or absence of an excess of cell elements in the fluid, and the nature of the cells which are present.

These different points may be considered in this order. Usually the cerebrospinal fluid escapes from the needle drop by drop, but in tuberculous meningitis the tension is generally increased and the fluid jets out in a continuous stream. In health it is a transparent, colourless, watery fluid, and, though it often retains this character in tuberculous meningitis, it may be greenish-yellow in colour or, in the last stage of the disease, even opaque. It thus differs from the fluid in cases of meningococcal, streptococcal, and pneumococcal disease, as in these conditions it is usually turbid or even purulent, though in chronic cases of meningococcal meningitis which recovered or became hydrocephalic Forbes [20] found that the cerebrospinal fluid was clear. On standing, a fibrinous clot frequently separates out, which is finer than the clot that appears in meningococcal or purulent meningitis.

The presence or absence of a reducing body in the fluid may give information of some value. Bernstein [5] found that Fehling's solution was reduced in twelve of fifty-three cases of tuberculous meningitis, even though the fluid was obtained only a few days before death; but that in cases of purulent meningitis the reducing body was invariably absent.

According to Forbes [21], who examined the cerebrospinal fluid from twenty-five cases of tuberculous meningitis, the reduction took place readily in nine, slightly in nine others, while in six it was absent.

On the other hand, in fifteen specimens of fluid from cases of meningococcal meningitis he found that two obtained in the acute stage readily reduced Fehling's solution, while six at a later stage of the disease only gave a partial reduction, and seven obtained during a chronic stage gave no reduction at all.

Formerly it was considered that the presence of sugar in a cerebrospinal fluid was sufficient in itself to exclude the diagnosis of an advanced tuberculous meningitis, but these observations prove that this is not the case. Bernstein suggests that the mistake arose through an insufficient quantity of cerebrospinal fluid being used, as ten cubic centimetres at least are required for the test.

The presence of tubercle bacilli may be detected by the microscope in one of two ways:—

(*a*) Where a clot is formed in the fluid the bacilli may be frequently found without difficulty by teasing it out on a slide and staining by the usual Ziehl-Nielsen method. In this way out of fifty-four specimens Forbes found bacilli in forty-three—*i.e.*, nearly 80 per cent.—while Bernstein found them in 100 out of 102 specimens examined; in some of the cases, however, the latter observer found it necessary to search for several hours before obtaining a successful result.

(*b*) By the examination of the centrifugalized cell deposit. This method is very tedious, as it requires a prolonged centrifugalization of the fluid and a search of several hours.

In cases where tubercle bacilli are not found by the methods just mentioned, proof of the tuberculous nature of the case may be obtained by the subcutaneous or intraperitoneal inoculation of guinea-pigs. This method, according to Bernstein, is absolutely trustworthy; on the other hand, Madame Hirschorn found that in patients suffering from nontuberculous meningitis, or from tubercle elsewhere than in the meninges, inoculation with the cerebrospinal fluid did not infect guinea-pigs.

The presence of tubercle bacilli may be also demonstrated by the inoculation of broth, glycerin-agar or potato-tubes, on which the bacillus grows fairly readily. This method, however, frequently fails, and a negative result is of no great importance.

When lumbar puncture was first commonly practised it was found that in tuberculous meningitis the bulk of the cells were lymphocytes, whereas in suppurative meningitis polymorphonuclear cells were markedly in excess. On the other hand, the cerebrospinal fluid of those patients who without actual disease of the meninges, presented the appearance of meningitis showed no increase of cell elements. From these facts it was hoped that a means had been discovered by which a definite diagnosis of meningitis and its nature could be made, but this unfortunately has been proved false by more recent investigations.

Rossi [48] states that the relationship between lymphocytosis and tuberculous meningitis is not constant, and that errors in diagnosis will occur if a pathognomonic value is attached to the presence of a lymphocytosis in the differential diagnosis of acute meningitis.

A constant predominance of mononuclear small cells was found by Goggia [24] in a case of meningococcal meningitis which terminated fatally.

Hutzner [40], again, concluded from the study of a series of cases confirmed by *post-mortem* examination—

(*a*) That in the cerebrospinal fluid of patients who are not suffering from tuberculous meningitis the cell elements present may be chiefly mononuclear, and (*b*) that there are cases of tuberculous meningitis in which an excess of polymorphonuclear cells occurs.

Porot's [43] results indicate that cyto-diagnosis is of little value, as in some cases all cellular reaction is absent, while in others the percentage of polymorphonuclear cells is very high.

Forbes [21], who has examined eighty cases of tuberculous meningitis, of which seventy were verified either by *post-mortem* examination or by the discovery of tubercle bacilli in the cerebrospinal fluid during life, found an excess of lymphocytes in fifty-one, an excess of polymorphonuclear cells in five, while in four cases the proportions of the cells were about equal. He considers from clinical and *post-mortem* evidence that the predominance of polymorphonuclear cells is associated with a chronic meningitis which terminates acutely, and in which several tuberculous nodules, in addition to a large amount of plastic lymph, are present. In cases of tuberculous tumours of the cerebrum and cerebellum without any accompanying meningitis, the same observer found that the cerebrospinal fluid was practically normal.

In 102 examinations of the cerebrospinal fluid made by Bernstein [5] only three exceptions were found to the mononuclear lymphocytosis usually present.

In this connection it is important to remember that a lymphocytosis is constantly or almost constantly present in the cerebrospinal fluids of patients suffering from para-syphilitic conditions, such as tabes dorsalis and general paralysis of the insane, and that this also occurs in some cases of general syphilitic disease of the central nervous system.

From these facts it is evident that a lymphocytosis in the cerebrospinal fluid of a case with the clinical symptoms of meningitis adds to the probability of its being tuberculous, but that a positive diagnosis cannot be made on these grounds alone.

To sum up briefly the results that may be obtained from the examination of the cerebrospinal fluid, we find that although the character, chemical constitution, and cytological contents are of considerable aid to the diagnosis of tuberculous meningitis, yet it can be only affirmed with certainty that a case of meningitis is tuberculous when one of the following conditions has been fulfilled:—

(1) When tubercle bacilli are discovered in the cerebrospinal fluid by the microscope, inoculation, or culture;

(2) When tubercle bacilli have been cultivated from the fluid;

(3) When inoculation of a guinea-pig has given a positive result.

Ophthalmoscopic examination of the fundus of the eye may sometimes afford valuable assistance, as in some cases of tuberculous meningitis tubercle of the choroid is present. Opinions differ widely as to the frequency with which this condition occurs in tuberculous meningitis. Treacher Collins [14] states that Bouchet found tubercle of the choroid in 10 per cent., Carpenter and Stephenson in 50 per cent. of unselected cases; while in contrast to these figures Jessop [34], who examined a large number of cases, found it present in approximately only 2 per cent. to 5 per cent., and then as a rule only shortly before death.

Accurate ophthalmoscopic examination of a patient suffering from meningitis is not easy, and errors in observation may occur, as syphilitic disease of the choroid sometimes presents an appearance closely resembling miliary tubercle, while plastic cyclitis with detachment of the retina may be mistaken for a conglomerate mass of tubercle.

In addition to these methods, one of the tuberculin reactions, or the estimation of the opsonic index of the blood, may be employed to confirm or refute the clinical diagnosis.

Finally, in cases in which a patient has died after a long remission of symptoms, or from another disease after recovery from the meningeal symptoms, the *post-mortem* examination may prove that the diagnosis of the tuberculous nature of the meningitis was correct.

The above methods of diagnosis may be summed up under two main headings:—

(a) Those which point to a meningitis as being of a tuberculous nature: (1) the clinical evidence; (2) the differential count of the cells in the cerebrospinal fluid obtained by lumbar puncture; (3) the presence of tubercle in the choroid on ophthalmoscopic examination; (4) tuberculin reactions.

(b) Those which definitely prove the tuberculous nature of the meningitis—namely: (1) The demonstration of tubercle bacilli in the cerebrospinal fluid by the microscope, by inoculation experiments, or by cultivation in artificial media; (2) *post-mortem* examination after remission or recovery.

III. A Critical Review of the Literature.

While the records of the London hospitals to which I have had access have failed to give me any cases of recovery from tuberculous meningitis in which the diagnosis was established beyond doubt, a search through the literature on the subject has afforded much evidence that patients with tuberculous meningitis may and actually do recover, and that in other cases remissions of the acute symptoms for several months may occur during the course of the disease.

The cases which I have collected from the literature on this subject may be conveniently divided into (1) those in which a long remission of the symptoms occurred, and (2) those which terminated in recovery. Each of these classes can be again subdivided into cases in which the tuberculous nature of the meningitis was proved definitely, and those in which there can be little doubt as to its tuberculous nature.

As in many of the cases that are classed under remissions the patients remained free from symptoms of active disease for months, it is evident that from the point of view of the subject of this paper there is no essential difference between these cases and those in which the recovery was permanent.

(1) *Remissions.*

(*a*) *Doubtful Cases.*—Don [16] records a case under the heading of "A Case of Tuberculous Meningitis in a Boy treated with Tuberculin. Recovery. Recurrence. Death." The diagnosis was made solely on clinical grounds, but the fact that after each injection of tuberculin the boy became worse for three days and then improved is mentioned as a confirmation of the diagnosis. The remission of symptoms was not for a prolonged period and no *post-mortem* examination was obtained so that the diagnosis is uncertain.

Hahn [27] reported a case in which the clinical symptoms were those of tuberculous meningitis, and in which there was a remission of the acute symptoms for three years. No *post-mortem* examination was made. The only further evidence produced as to the nature of the disease was the fact that there was a tuberculous family history. Consequently the proof cannot be considered conclusive.

Dudon [17] reported a case to which Carrière and Lhôte [11] refer. In a boy of 17 an attack, in which for three weeks symptoms of meningitis were present, abated sufficiently for the boy to return to work. But an alteration in the lad's character was noted, and he suffered from tinnitus and occipital headache. A second and similar attack occurred three and a half years later, leaving severe pains in the head after recovery. The boy then became a drunkard, and died after six months in a final attack with symptoms of meningeal inflammation. No *post-mortem* examination was made. The criticism passed on the case when it was first reported was that the first attack was possibly one of enteric fever, and that the change of character which followed it was due to alcoholic excess. There is certainly no proof in this case of the nature of the meningitis.

Coulon [13] and Sepet [52] each observed a case in which the family history was very bad, the parents and other near relations being tuberculous, while the patients themselves suffered from tuberculosis. In Coulon's case death resulted from an acute miliary tuberculosis which had been preceded by two remissions of meningeal symptoms at intervals of twelve months and of four and a half years respectively. In Sepet's case the remission lasted a year. The presence of tuberculous lesions in other organs, with a history of tuberculosis in near relations, points to the probability of the meningitis in these cases being tuberculous, but it does not prove definitely that such was the case.

(*b*) *Certain Remissions.*—In the following cases of remission there can be no doubt as to the nature of the case.

Carrière and Lhôte [11] report four cases which came under their own observation and review the cases which had been previously recorded.

In their first case a boy, aged 5 years, was observed with Jacksonian epilepsy that began in the left side of the body and rapidly became general. Lumbar puncture showed a marked lymphocytosis in the cerebrospinal fluid; this demonstrated the presence of meningitis, as in cases of tuberculous tumours of the brain the cerebrospinal fluid obtained by lumbar puncture presents the features of healthy cerebrospinal fluid with but a very slight cell deposit. The tuberculous nature of the case was proved by the inoculation of a guinea-pig with a positive result. After four months, during which he suffered from two attacks of Jacksonian epilepsy and vomiting, the lad was able to leave the hospital. But nine and a half months later he was readmitted with symptoms similar to those of his previous attacks.

The lack of a *post-mortem* examination prevented the most satisfactory proof of the condition of the brain and meninges, but the positive result of the inoculation of the guinea-pig, together with the examination of the cerebrospinal fluid, enables us to consider the first attack of Jacksonian epilepsy as due to a tuberculous lesion in the cerebral cortex, with a meningitis that became latent for a period of some ten months.

The second case was that of a boy, 7 years old, in whose family there was a history of tuberculosis, while he himself had tuberculous enlargement of the cervical glands, one of which had broken down and formed a cold abscess. There was the clinical picture of tuberculous meningitis, and the diagnosis was confirmed by definite lymphocytosis of the cerebrospinal fluid, and by the death of a guinea-pig from tuberculosis after inoculation with 20 c.c. of the cerebrospinal fluid.

Improvement began ten days after admission, and at the end of two months the only physical signs remaining were slight strabismus and slight nystagmus when the eyes were in the extreme lateral position, together with a sluggish reaction of the pupils to light.

After an interval of four months a recurrence took place and the patient died. No *post-mortem* examination was made.

Their third case was that of a boy, aged 6½, whose mother was tuberculous; two brothers had died from meningitis and a sister was scrofulous. He himself had had a suppurating cervical gland when four years old.

For the first few days the diagnosis was doubtful, but later definite symptoms of meningitis appeared, and an examination of the cerebrospinal fluid showed a definite lymphocytosis. Moreover, a guinea-pig was inoculated and died of general peritoneal tuberculosis.

The discovery of tubercle bacilli in the cerebrospinal fluid, obtained by lumbar puncture at a later date, further confirmed the diagnosis; but, instead of death terminating the illness, the boy improved and gradually returned to almost perfect health: constipation, slight facial paralysis on the right side, and rapidity in respiration were the only physical signs remaining two months after the onset of the disease. The boy remained well until nine months later, when death occurred in a recurrence of the symptoms. A *post-mortem* examination was not allowed.

The last case reported by these authors was that of a boy, aged 4, in whom the clinical symptoms, the presence of a definite lymphocytosis in the cerebrospinal fluid, and the death of a guinea-pig from tuberculosis after inoculation with the fluid, made the diagnosis of tuberculous meningitis certain. A discrete tubercle of the choroid was also discovered in one eye on ophthalmoscopic examination. Improvement began on the eighth day after admission and continued until the child was discharged two months later. He was then irritable, constipated, and suffered from slight twitchings of the eyelids from time to time. There was also some weakness with slight tremor of the right arm. The respirations in this case also were rapid, being thirty in the minute. Ophthalmoscopic examination two months after his discharge from hospital showed that the tubercle of the choroid had vanished, but that there was now a diffuse irido-choroiditis, with slight œdema of the optic discs.

Four months after being discharged from hospital the child was readmitted with a recurrence of similar symptoms, and death rapidly ensued. On *post-mortem* examination definite signs of old meningitis were found; there was a dense sclerosis of the meninges over the fissure of Rolando on the left side, with general disseminated tuberculosis. Carrière and Lhôte [11] concluded from the *post-mortem* examination that there was a plaque of old sclerosed tuberculous meningitis over the left Rolandic fissure, with a recent miliary tuberculosis around it.

A case published by Rilliet [46] in 1853, and referred to by Carrière and Lhôte in their paper, is well worthy of comparison with the case just mentioned.

A boy, aged 5½, presented the clinical symptoms of tuberculous meningitis, but, instead of dying, he began to improve after fourteen days, and eventually recovered. Some time later he fell from a height and sustained a fracture of the base of his skull, from which he recovered and was able to run about; no trace of either the illness or the accident remained, and for five and half years the child continued in excellent health, with such perfect intellectual development that he was able to keep the accounts of his father, who was an innkeeper. At the end of that time a recurrence of his former illness took place, and he died in ten days. *Post-mortem* examination revealed a mass of old meningitis over the middle and outer lobes of the left hemisphere of the brain, while a recent disseminated tuberculosis, of the type usual in tuberculous meningitis, was present in the meninges, especially at the base of the brain. The site of the old meningitis was very similar to that found by Carrière and Lhôte in the only case in which they were able to make a *post-mortem* examination.

The following case is of much interest. In October, 1900, a child, aged 8, was admitted under Rocaz [47] into the Children's Hospital, Bordeaux, with the characteristic clinical symptoms of tuberculous meningitis.

The lymphocytosis observed on examination of the cerebrospinal fluid, together with a history of tuberculosis in the parents and signs of phthisis in the patient, added to the probability that the meningitis was tuberculous. The child, however, recovered, the lymphocytosis disappeared, and, as at the end of nine months no signs of ill-health were detected, Rocaz published the case as an apparent recovery after an attack of tuberculous meningitis.

The sequel was published by Renée Cruchet [15], under whom the child was readmitted twenty-two months later with symptoms similar to those of the first attack, with, in addition, a marked deviation of the tongue to the right. Two months after readmission the child had improved to such an extent that he was up, playing with the other convalescents in the ward; but on this occasion the recovery did not last any length of time, and death followed a recrudescence of the disease. *Post-mortem* examination removed all doubts of the correctness of the diagnosis, as a thick mass of old tuberculous meningitis was found in the region of the pons Varolii, while recent miliary tubercles studded the meninges at the base of the brain.

Hermann [38] also published a case of remission of the symptoms for four months in a child who died during the second attack. *Post-mortem* examination showed that the meninges at the base of the brain were matted together by an exudation which extended into the Sylvian fissure, and in which, on microscopical examination, typical perivascular miliary tubercles were found.

(2) *Recoveries.*

(*a*) *Doubtful Cases.*—Buchanan [8] showed a child before the Liverpool Medical Institute who had had the clinical symptoms of tuberculous meningitis, in whom Calmette's ophthalmo-reaction gave a positive result, while there was a lymphocytosis of the cerebrospinal fluid withdrawn by lumbar puncture. Improvement followed an inoculation with $\frac{1}{1000}$ mg. of new tuberculin; this inoculation was repeated three weeks later, and uninterrupted recovery ensued. In this case the positive result of Calmette's ophthalmo-reaction shows the probable presence of tubercle somewhere in the body, while the lymphocytosis found in the cerebrospinal fluid is greatly in favour of the tuberculous nature of the meningitis; but on these facts alone the definite assertion that the meningitis was tuberculous cannot be made.

Jirasek [35] has reported a case in which the family history was very bad, the parents and other near relations being tuberculous, while the patient herself had pulmonary tuberculosis. Recovery took place after two attacks of meningeal symptoms with a year's interval between them.

Ovazza [42] and Porot [43] each record a case of recovery which clinically presented the typical appearance of tuberculous meningitis, but no further confirmation is given as to the nature of the disease. An interesting fact is recorded by Porot [43]: it is that there had been an attack of meningitis previous to the one he himself observed, which had left nystagmus, strabismus, and a facial tic. In this case the patient had an extremely bad family history as regards tubercle.

In the case recorded by Troisier and Broulé [59] the clinical symptoms of tuberculous meningitis were accompanied by a marked lymphocytosis in the cerebrospinal fluid, but no pathogenic organisms could be discovered. The meningeal symptoms lasted only eight days, and recovery was perfect, no sequelæ remaining after the meningeal symptoms had disappeared. Even the lymphocytosis was no longer present in the fluid obtained by a lumbar puncture done during convalescence. These authors regarded the presence of a lymphocytosis as conclusive evidence of the tuberculous nature of the meningitis, but it must be remembered that Goggia [24] found a constant predominance of lymphocytes in the cerebrospinal fluid in a fatal case of meningococcal meningitis during the course of the disease.

In cases reported by Sepet [52] and by Firmin Carles [9] it is recorded in confirmation of the clinical diagnosis that tuberculosis existed in near relations, that the progress of the disease was similar to that of tuberculous meningitis, and that positive results were obtained, in Sepet's case with the sero-reaction of Arloing and Commont, in Carles' with Calmette's ophthalmo-reaction. The examination of the cerebrospinal fluid also revealed a lymphocytosis, which was considered to be definite proof of the tuberculous nature of the disease. The positive results of the tuberculin tests may have been due to tuberculosis in other parts of the body—for example, the mesenteric or posterior mediastinal glands. Further, in Carles' case measles, followed by whooping-cough and broncho-pneumonia, occurred during the presence of the meningeal symptoms, and, even though the measles did not appear until a month after the onset of the illness, the fact that meningeal symptoms often usher in an infectious disease must not be forgotten. The case must be consequently received with caution.

Senger [51] has stated that five cases in which he made the diagnosis of tuberculous meningitis had recovered when treated with mercurial inunction, and that in three of them the recovery remained permanent; but he admitted that, as tubercle bacilli had not been demonstrated in the cerebrospinal fluid, the diagnosis was uncertain. He argued that the slow onset, the photophobia, and the general condition of the patients greatly favoured the diagnosis of tuberculous rather than of acute cerebrospinal meningitis.

Tripier [58] in 1902 also recorded eight cases of tuberculous meningitis which he had cured in the prodromal stage by repeated blistering of the head. As, however, he stated in this paper, that the treatment to be successful must be applied as soon as meningitis is suspected, and that it is too late if the least trace of contracture or of paralysis has appeared, his cases probably were not tuberculous meningitis.

Law [44], again, in a lecture delivered at the Medical Graduates' College and Polyclinic, said: "In two cases of apparent tuberculous meningitis, with all the classical signs, the symptoms entirely disappeared after four injections of tuberculin, and both children made rapid recovery. In other two instances tuberculin had no effect, and the children died. Of course, it is impossible to be sure that the first two cases were of a tuberculous nature, as tubercle bacilli could not be recovered by lumbar puncture, but I have no doubt myself that they were."

(b) *Undoubted Cases.*—In the following cases, on the other hand, the correctness of the diagnosis is, I think, indisputable.

Freyhan [22], who was the first to publish a case in which the diagnosis was confirmed during life by the demonstration of tubercle bacilli in the cerebrospinal fluid, recorded the case of a workman, aged 20, who was admitted to hospital, having suffered for three days with very severe headache in the occipital region, pains in the legs, and insomnia.

Examination revealed the characteristic symptoms of meningitis, which rapidly proceeded to the stage of coma. The diagnosis of cerebrospinal meningitis was at once made, but was altered to tuberculous meningitis when tubercle bacilli were found in the cerebrospinal fluid removed by lumbar puncture. A very unfavourable prognosis was given; but after the tenth day the patient steadily improved, and in three weeks was able to leave his bed. Three months later the man was well, though the optic neuritis which was present on admission persisted.

Henkel [29] published the case of a boy, aged 10, who suffered from meningitis with double optic neuritis. Lumbar puncture and an examination of the cerebrospinal fluid disclosed numbers of tubercle bacilli, thus proving the case to be tuberculous meningitis. The boy remained in a serious condition for twelve days, and then gradually improved; a month after the onset of the attack he was able to walk. As the boy at this period was unsteady on his feet and reeled to the right, it was thought that there might be a solitary tuberculous mass in the cerebellum; but all these symptoms disappeared within three weeks, and the boy was discharged without any trace of his illness.

Gross [26] records the case of a boy, aged 17, who had been struck on the head with a beer-bottle five days before he came under observation. The discovery of tubercle bacilli in the fluid withdrawn by lumbar puncture proved that the case was one of tuberculous meningitis and that the original diagnosis of serous meningitis was wrong. The boy gradually improved and made a perfect recovery from the meningeal disease, but twelve months after the onset of the meningitis he was found to have pulmonary tuberculosis. An interesting point in this case was that the differential count of the cell elements present in the cerebrospinal fluid, in exception to the general rule, showed a predominance of polymorphonuclear cells.

A very similar case is reported by Stark [53]. The diagnosis of enteric fever was at first made, as the patient, a man aged 44, was suffering from fever, vomiting, headache, diarrhœa, and retraction of the head. After four days, however, the appearance of ocular phenomena—viz., optic neuritis and inequality of pupils—together with a paresis of the face, showed that the disease was a meningitis. Lumbar puncture was performed, and tubercle bacilli were found in the cerebrospinal fluid. The patient recovered completely and increased some 3 lb. in weight while under observation.

Barth [4] again published the case of a girl, aged 32 months, in which the symptoms made him consider the possibility of tuberculous meningitis. As the disease progressed the symptoms became more characteristic, and the diagnosis was confirmed by the discovery of tubercle bacilli in the cerebrospinal fluid. Contrary to expectation, the child lived and, after an illness of six months' duration, was restored to health.

At the International Congress of Tuberculosis held in Washington, during 1908, Stiles [54] related two cases which showed the possibilities of surgical treatment in this affection. In both cases an operation was performed to relieve intracranial pressure, and in the fluid which escaped on opening the dura mater tubercle bacilli were found. The cases both recovered; in one a recurrence of symptoms followed the closure of the wound, but disappeared when the wound was reopened. Stiles stated that in his opinion many lives would be saved if the above surgical procedure were adopted more frequently.

The following is an abstract of a case reported by Alanzino [1]: The patient presented the clinical symptoms of tuberculous meningitis; the cerebrospinal fluid gave a cell deposit in which lymphocytes were greatly in excess, as is usual in this disease, and a guinea-pig inoculated with some of the fluid showed tuberculous lesions when killed on the fiftieth day. The child recovered and was in perfect health during the six months that he remained under observation.

Tedeschi [56] observed a case of recovery from tuberculous meningitis in a girl aged 14. The differential count of the cell-elements in the cerebrospinal fluid, together with the clinical symptoms, pointed to tuberculous meningitis, and this diagnosis was confirmed by the positive result obtained from the inoculation of a guinea-pig. The patient recovered and was helping her mother in household duties when last seen. Interesting points in this case were the absence of fever and of Kernig's sign during the illness, as well as the almost complete disappearance of the lymphocytosis present in the cerebrospinal fluid prior to her discharge from hospital.

A case is recorded by Jemma [33] in which a boy, aged 3½, suffered from an illness that was proved to be tuberculous meningitis by the clinical symptoms, the presence of a lymphocytosis in the cerebrospinal fluid, and the inoculation of a guinea-pig with a positive result. The convalescence was prolonged, but three years later the child was in good health.

Gareiso [23] records a case from the Argentine Republic of a boy, aged 8, in whom the onset was slow and the symptoms typical. The cerebrospinal fluid on examination showed the lymphocytosis usual in tuberculous meningitis, and inoculation of a guinea-pig resulted in its death from tuberculosis. The child was quite well at the end of six months and continued in good health for at least twelve months after the onset of the attack.

Claisse and Abrami [12] showed a case of recovery before the Société Médicale des Hôpitaux de Paris. The onset of the illness in the patient, a man aged 30, was characteristic and was accompanied by fatigue, headache, loss of flesh, and insomnia. Later the patient suffered from delirium, accompanied by violent actions, ideas of persecution, anorexia, obstinate constipation; the pupils were unequal and reacted slowly to light. There was a lymphocytosis of the cerebrospinal fluid and a guinea-pig which was inoculated died of tuberculosis. The patient made a satisfactory recovery.

In a discussion which took place Montard-Martin, Sicard, and Achard mentioned cases under their care in which remissions had taken place, while Dufour stated that on two occasions he had made *post-mortem* examinations and had found cerebral and meningeal tubercles which were not of the same age.

At the next meeting of the same Society Vaquez and Digne [60] communicated a similar case of recovery from tuberculous meningitis. The clinical signs were most characteristic, and the diagnosis was confirmed by the examination of the cerebrospinal fluid, as well as by the positive result obtained from the inoculation of it into a guinea-pig. After recovery, instead of the large number previously present, the cerebrospinal fluid contained but few lymphocytes and no longer produced tuberculous lesions in guinea-pigs.

Vaquez and Digne considered that meningitis was undoubtedly present unless the clinical symptoms, as well as the macroscopic and microscopic character of the cerebrospinal fluid, were of no significance, and that the tuberculous nature of the meningitis was conclusively proved by the fact that the liquid withdrawn by lumbar puncture caused tuberculosis in a guinea-pig.

In a case recorded by Rossini [49] the tuberculous nature of the disease was demonstrated by the inoculation of a guinea-pig and the production of a generalized tuberculosis in the animal. There was complete recovery.

The case recorded by Janssen [31] was that of a soldier, aged 19, who, after an attack which was diagnosed as tuberculous meningitis, recovered sufficiently to resume his military duties. Three years later the man died from phthisis after an illness of four months. *Post-mortem* examination revealed yellow caseous masses on either side of the superior longitudinal sinus, which on careful examination were found to consist of small round tubercles. Scattered tubercles, completely obsolete, were also found over the pia mater, especially along the course of the vessels and in the sulci between the convolutions. Similar obsolete tubercles were found around the optic chiasma at the base of the brain. Microscopical examination failed to disclose tubercle bacilli and indicated that the tubercles were of considerable age.

Janssen refers to cases of recovery from tuberculous meningitis which I have been unable to trace. Politzer observed a case in which death occurred in a relapse three years after the first attack. On *post-mortem* examination he found thick fibrous tissue over the base of the brain, which was evidently produced by the previous attack, and young disseminated tubercles in the pia-arachnoid. Schwalbe observed two cases in which recovery from tuberculous meningitis took place. The patients died later from diphtheria, and the diagnosis was then confirmed by the discovery of a meningeal tuberculosis not of recent date. Another case of recovery from tuberculous meningitis is reported by Carrington, in which diagnosis was confirmed by *post-mortem* examination as the patient died later from psoas abscess. Finally, Janssen refers to a paper by Leube, in which there is reported the case of a girl, aged 24, who presented signs of phthisis and of inflammation of the spinal meninges. The latter symptoms subsided, leaving only a few traces behind. The diagnosis of tuberculous spinal meningitis was later confirmed, as the girl died from generalized tuberculous meningitis, and *post-mortem* examination revealed an older tuberculous affection of the spinal meninges.

A case of recovery from tuberculous meningitis was published by Riebold [45]. A young girl, aged 6, presented all its symptoms; the diagnosis was placed beyond doubt by the discovery of numerous tubercle bacilli in the cerebrospinal fluid and the intraperitoneal inoculation of guinea-pigs, with as a result the production of typical generalized tuberculosis. The treatment of this case was lumbar puncture repeated daily until 574 c.c. of fluid had been withdrawn. After the early punctures improvement took place and the general condition gradually returned to normal. At the beginning of 1906, two months after the onset of the illness, the patient was considered out of danger, and when seen in August of the same year was in excellent health. The evidence as to the tuberculous nature of the meningitis is in this case very conclusive.

Possibly the most interesting case of all is that recorded by Rumpel [50], who exhibited at the Aerztlicher Verein in Hamburg the brain of a boy, aged 17, who had died of phthisis. At the age of nine the lad had an acute attack of meningitis in which there was retraction of the head, hyperæsthesia, absence of the knee-jerks, bilateral optic neuritis, ptosis and, paralysis of the external recti. Lumbar puncture gave a fluid in which tubercle bacilli were found. The boy, who was treated by repeated lumbar punctures, recovered, and, though for some months he was dull and stupid, he eventually returned to his normal mental condition and was able to keep pace with others of his age at school, while all abnormal physical signs had disappeared. He remained about four years in good health, but then developed a tuberculous abscess in the axilla, which was followed by pulmonary tuberculosis; death took place eight years after the onset of his meningeal symptoms. At the *post-mortem* examination it was found that the membranes over the fissure of Sylvius on either side of the brain were thickened and glued together, but miliary tubercles were no longer visible. In this case not only was there confirmation of the diagnosis during life by the discovery of tubercle bacilli, but also a *post-mortem* examination in which the site of the old meningitis was discovered.

In the three cases which follow the observers record the discovery of tubercles in the choroid on ophthalmoscopic examination as a confirmation of the diagnosis of tuberculous meningitis.

In Lunn's [37] case the patient was a girl, aged 6, whose father had died from phthisis; she was admitted to the St. Marylebone Infirmary with symptoms resembling those of enteric fever. Six days after admission the symptoms became those of meningitis, and there was a blood-stained discharge from the left ear, which, however, soon ceased. On ophthalmoscopic examination optic neuritis was discovered in the eyes, and in the left a large white oval area about one-third the size of the optic disc was seen. This area appeared somewhat raised and was free from pigment, except for a fine line at the lower border. The retinal vessels passed over it. Two smaller patches were also noticed: the one, white in colour, was situated near the lower and outer edge of the large area; the other, of a greyish hue, was placed above the inferior temporal vessel. The eyes were examined by Lunn, Rayner-Batten, and Lawford, who agreed that tubercle of the choroid was present, and accordingly a diagnosis of tuberculous meningitis was made. Three days later the child began to improve and recovery ensued. Before the child left the Infirmary the optic neuritis and one of the smaller patches had vanished. The large oval area was still present, and the small patch which remained was fading.

Although the symptoms in this case were not very conclusive, the presence of the optic neuritis and other cerebral symptoms makes the diagnosis of meningitis probable. The presence of tubercles of the choroid points to the meningeal disease being of a tuberculous nature.

Dujardin-Beaumetz [18] recorded a case in which the symptoms were at first supposed to be those of enteric fever, but gradually progressed until the typical appearance of tuberculous meningitis was present. The man, however, recovered and was discharged without any trace of the illness. Dujardin-Beaumetz, feeling sure of his diagnosis, had an examination made by an ophthalmic surgeon, who found in the right eye not only all the signs which are characteristic of tuberculous meningitis, but also a tubercle of the choroid, which he believed confirmed the diagnosis.

In Thomalla's [57] patient the parents were tuberculous, and the man himself suffered from rectal fistulæ of a tuberculous nature. The symptoms were those of tuberculous meningitis, and an ophthalmoscopic examination disclosed two absolutely typical recent tubercles in the choroid of the left eye. The patient made an excellent recovery, and three months after the onset of the attack the choroidal tubercles were found to have disappeared, leaving only a pigmented scar behind.

IV. Differential Diagnosis.

In the differential diagnosis of tubercular meningitis there must be taken into consideration that condition known as *meningism*, a term applied to the appearance of symptoms which simulate more or less closely those of meningitis in the course of infective illnesses and intoxications, but no morbid lesions can be found in the meninges after death; and *serous meningitis*. This condition, in which there is an exudation of serous fluid into the subarachnoid space, can also produce the symptoms of an infective meningitis; but the temperature is usually not as high as in true meningitis, and it soon returns to the normal, the headache is less severe, the retraction of the head is less marked, and the physical signs are only transitory. Optic neuritis is frequently present and the knee-jerks may be absent.

These conditions can be, however, distinguished from true meningitis by lumbar puncture, as the fluid, though often under great tension, is clear, contains an increased amount of albumin, but only a few cell elements and no micro-organisms.

Serous meningitis is usually due to some toxin circulating in the blood, and it is of interest in relation to our subject that many authors, as Münzer [39], Riebold [45], and Biedert [7] consider that the tubercular toxines occupy the most important place in its ætiology.

In addition to the clinical evidence of this we have the experimental work of Armand-Delille [2]. By the injection of extracts of toxins obtained from the tubercle bacillus into the subarachnoid space of the brain he was able to produce a diffuse infiltration of the pia mater with mononuclear and polymorphonuclear leucocytes, which in places tended to form nodules, in the centre of which caseation could be often observed; similar cells also infiltrated the perivascular spaces, and secondary changes were often observed in the adjacent nerve cells. In a later stage these changes were represented by a sclerosis of the pia-arachnoid. The lesions produced by the local action of tuberculous toxins thus resemble closely those of tuberculous meningitis.

Laignel-Lavastine [36] has also shown that pathological changes can be detected in the nerve cells of the cerebral cortex in general tuberculosis, even when there is no infection of the brain.

These clinical and experimental facts demonstrate that the tuberculous toxins can produce lesions both of the meninges and of the cerebral cortex apart from direct infection.

In *serous*, however, as distinguished from *infective* meningitis the exudation is caused solely by the action of toxins, and *micro-organisms cannot be found in the cerebrospinal fluid.*

From this fact it follows that the cases I have collected, in which the presence of tubercle bacilli has been demonstrated in the cerebrospinal fluid either microscopically or by inoculation, must be regarded as recoveries from true tuberculous meningitis; but in those cases in which the diagnosis has been based on the clinical aspect alone, or on this together with the presence of a lymphocytosis of the cerebrospinal fluid and a positive result given by tuberculin reactions, it is difficult to say whether there has been a serous or a true tuberculous meningitis.

It is well known that the progress of tuberculosis in other organs may be interrupted by periods of quiescence, or even by recovery. Apparently the resistance of the individual to the disease is an important factor, and that this comes into play in tuberculous meningitis is evident from the course and duration of the disease in different individuals. Hall and Hopkins [28], for instance, found that the duration of the disease varied between three and ninety days in fifty-two cases which they observed.

Despite the clinical experience of many expert observers and the statements to the contrary contained in many medical text-books, it is therefore not irrational to consider the possibility that tuberculous meningitis, too, may terminate in recovery or in long periods of remission.

In the *post-mortem* examinations which have been made on patients in whom remissions or recoveries have occurred the earlier tuberculous lesions have been represented merely by a sclerosis of the meninges; we may consequently assume, in those cases which recover from tuberculous meningitis, either that the virulence of the bacilli has been so small that the lesion has had little or no tendency to become disseminated over the meninges, or that the resisting power of the patient has been so great that the tuberculous meningitis terminated at an early period of its existence.

The fact that a recurrence of the disease has taken place after many years (in Rilliet's [46] case five and a half years intervened between the attacks) proves that the localized form is a constant source of danger, and that the possibility of a recurrence must be always considered.

V. Treatment.

It is interesting to review the treatment adopted in the cases which recovered, or in which there was a long period of remission from active disease. I hoped to find some one method which might be considered as contributing largely to the successful termination, but this has not proved the case, for the methods of treatment adopted differ widely, and each physician attributes the fortunate termination to the particular method he employed.

Janssen [31] considered that the recovery of the patient under his care was due to enormous doses of iodide of potassium, 600 gr. a day being the amount administered. Barth [4], on the other hand, obtained no improvement with potassium iodide, baths and cold effusions to the head, but attributed the recovery in his case to the abstraction of blood by leeches applied over the mastoid processes for eight consecutive days.

Rumpel [50] and Riebold [45] believed that recovery in their respective cases was due to repeated lumbar puncture, which relieved tension and withdrew the toxins present in the cerebrospinal fluid. Stiles [54] strongly recommended operation with the same object in view. Finally, Rossini [49] considered the recovery of his case due to the injection of 1 oz. of a 1 per cent. solution of iodoform in almond oil into the subarachnoid-space every six hours, and every second day an intravenous injection of a 1 per cent. solution of iodine and potassium iodide.

As successful results have been obtained by methods which differ so widely, and as each of these methods has been tried repeatedly in other cases without success, the only safe conclusion is that the treatment employed had little or nothing to do with the recovery of the patients, and that the fortunate termination depended either on the increased resistance offered by the patients to the action of the tubercle bacilli, or on the small virulence of the bacilli themselves.

Finally, I must take this opportunity of expressing my thanks to Dr. F. E. Batten, who suggested this investigation to me and aided me in it with his advice and criticisms.

VI. CONCLUSIONS.

The conclusions to be drawn from the above facts are :—

(1) That undoubtedly long remissions and even recoveries do occur in tuberculous meningitis.

(2) That recoveries are possibly more frequent than has been believed, since no fewer than twenty undoubted cases have been recorded since 1894, while other cases of recovery have been published in which the same definite proof of the nature of the disease has not been afforded, but some of which probably were true cases of tuberculous meningitis.

(3) That in these cases either the resistance of the individual is so much greater than usual that the disease is checked early in its course, or the virulence of the bacilli is so much less than usual that the lesion in the meninges becomes localized and later undergoes a fibrous change.

(4) That the lesion in the meninges may at a later period form the focus of a fresh infection which usually terminates fatally, and that consequently the prognosis in these cases must be guarded.

(5) That no treatment up to the present has been discovered which has had any specific effect in promoting the favourable termination of the disease.

References.

[1] Alanzino. *Riformi med.*, 1903, Ann. xix, p. 936 (abs. *Rev. neurol.*, 1903).

[2] Armand-Delille. Thèse de Paris, 1903.

[3] Barlow, Sir Thomas. "Clifford Allbutt's System of Medicine," 1899. 1st ed., vol. vii., p. 481.

[4] Barth. *Münch. med. Wochenschr.*, 1902, Bd. xlix, S. 877.

[5] Bernstein. *Med. News*, 1905., vol. lxxxvi, p. 1105.

[6] *Idem.* *Mount Sinai Hosp. Reports*, 1907.

[7] Biedert (*cit.* Oppenheim).

[8] Buchanan. *Lancet*, 1907, vol. ii, p. 1764.

[9] Carles. *Arch. de Méd. des Enfants*, 1908, t. xi, p. 130.

[10] Carpenter. *Reports of the Soc. for the Study of Disease in Children*, 1902, vol. ii, p. 203.

[11] Carrière et Lhôte. *Rev. de Méd.*, 1905, t. xxv, p. 469.

[12] Claisse et Abrami. *Gaz. des Hôp.*, 1905, t. lxxviii, p. 667.

[13] Coulon. *Méd. enfantile*, 1894, t. i, p. 340 (*cit.* Carrière et Lhôte).

[14] Collins, E. Treacher. *The Ophthalmoscope*, 1907, vol. v, p. 2.

[15] Cruchet. *Rev. neurol.*, 1902, t. x, p. 1077.

[16] Don. *Brit. Med. Journ.*, 1907, vol. i, p. 1360.

[17] Dudon. *Mém. et Bull. de la Soc. de Méd. de Bordeaux*, 1876 (*cit.* Carrière et Lhôte).

[18] Dujardin-Beaumetz. *Bull. de la Soc. de Méd. des Hôp. de Paris*, 1878.

[19] Emery. "Clinical Bacteriology and Hæmatology," London, 1908.

[20] Forbes. *Quarterly Journ. of Med.*, 1908, vol. i, p. 109.

[21] *Idem.* *Lavori e Riviste di Chimica e Microscopia Clinica*, 1908., vol. i.

[22] Freyhan. *Deutsche med. Wochenschr.*, 1894, Bd. xx, S. 707.

[23] Gareiso. *Revista de la Soc. med. Argentina*, 1908 (abs. *Arch. de Med. des Enfants*, 1908).

[24] Goggia. *Gaz. degli Ospedali e delle Clin.*, 1905, Ann. xxvi, p. 133 (abs. *Rev. neurol.*, 1905).

[25] Gowers. "A Manual of Diseases of the Nervous System," London, vol. ii.

[26] Gross. *Berliner klin. Wochenschr.*, 1902, Bd. xxxix, S. 776.

[27] Hahn (*cit.* Carrière et Lhôte).

[28] Hall and Hopkins. *Journ. Nerv. and Ment. Diseases*, 1906, vol. xxxiii, p. 242.

[29] Henkel. *Münch. med. Wochenschr.*, 1900, Bd. xlvii, S. 799.

[30] Hirschorn. Thèse de Paris, 1903.

[31] Janssen. *Deutsche med. Wochenschr.*, 1896, Bd. xxii, S. 169.

[33] Jemma. *La Pediatria*, 1907 (abs. *Rev. neurol.* 1908).

[34] Jessop. *St. Bartholomew's Hosp. Reports*, 1905, vol. xli, p. 183.

[35] Jirasek. *Rev. v. Neurologii*, 1904 (abs. *Rev. neurol.*, 1905).

[36] Laignel-Lavastine. *Rev. de Méd.*, 1906, t. xxvi, p. 270.

[37] Lunn. *Trans. Ophthalmol. Soc. London*, 1904, vol. xxiv, p. 127.

[38] Mermann. *Beitrage zur klin. Chirurg.*, 1902, Bd. xxxiv, S. 268.

[39] Munzer. *Prager med. Wochenschr.*, 1899, No 46.

[40] Hutzner. *Arch. gén. de Med.*, 1901, t. clxxxviii, p. 345.

[41] Oppenheim. "Lehrbuch der Nervenkrankheiten," Wien, 5te Aufl., 1908.

[42] Ovazza. *Riforma med.*, 1906, vol. xxii, p. 964.

[43] Porot. *Rev. de Méd.*, 1908, t. xxviii, p. 37.

[44] Raw. *Lancet*, 1908, vol. i, p. 481.

[45] Riebold. *Münch. med. Wochenschr.*, 1906, Bd. liii, S. 1709.

[46] Rilliet. *Bull. de la Soc. de Méd. des Hôp. de Paris*, 1853 (*cit.* Carrière et Lhôte).

[47] Rocaz. *Arch. de Méd. des Enfants*, 1901, t. iv, p. 738.

[48] Rossi. *Gaz. degli Ospedali e delle Cliniche*, 1905, Ann. xxvi, p. 473.

[49] Rossini. *La Clinica mod.*, 1904, vol x, p. 445 (abs. *Rev. neurol.* 1905).

[50] Rumpel *Deutsche med. Wochenschr.*, 1907, Bd. xxxiii, S. 2021.

[51] Saenger. *Neurol. Centralb.*, 1903, Bd. xxii, S. 794

[52] SEPET. *Médecine mod.*, 1902, t. xiii, p. 225.

[53] STARK. *Neurol. Centralb.*, 1908, Bd. xxvii, S. 633.

[54] STILES. *Proc. Internat. Cong. of Tuberculosis*, Washington, 1908.

[55] TAYLOR, F. "Manual of the Practice of Medicine," London, 1908.

[56] TEDESCHI. *Gaz. degli Ospedali e delle Cliniche*, 1905, vol. xxvi, p. 1045 (abs. *Rev. neurol.*, 1906).

[57] THOMALLA. *Berliner klin. Wochenschr.*, 1902, Bd. xxxix, S. 565.

[58] TRIPIER. *Provence médicale*, 1902 (abs. *Rev. neurol.* 1903).

[59] TROISIER et BRULÉ. *Rev. neurol.*, 1907, t xv, p 1135

[30] VAQUEZ et DIGNE. *Gaz. des Hôp.*, 1905, t. lxxviii, p. 702

Publications Recently Received.

[*Notes on a book under this heading do not preclude a subsequent review.*]

Arbeiten aus der deutschen psychiatrischen Universitäts-Klinik in Prag. Herausgegeben von Professor Dr. Arnold Pick. Mit zahlreichen Abbildungen im Text und 11 Tafeln, S. 143. Berlin: Karger, 1908.

One of the most interesting papers in this collection is that on a case where the knowledge of the position of parts of the body was gravely disturbed. The patient did not know where such parts as her ears, her eyes, her legs were to be found. Told to touch her nose, she could do so automatically; but if her hands were held for a moment, so that this automatic response was prevented, she searched in her lap and other parts of her body. A full abstract is given of Professor Pick's contribution to the Amsterdam Congress on asymboly and aphasia. Papers on tumours of the hypophysis, on the symptomatology produced by atrophy of the occipital lobes, on local senile atrophy of the brain, on the loss of cortical fibres in general paralysis, make up an interesting volume.

Ueber die Functionen von Hirn und Rückenmark. (Gesammelte Mitteilungen—Neue Folge.) Von Hermann Munk. Mit 4 Textfiguren, S. 371. Berlin: Hirschwald, 1909.

We welcome this reprint of the papers of Professor Munk on the functions of the cortex and the cerebellum. For when the dust of a controversy has died down it is well to look back on the documents to learn how the universal beliefs of to-day have been built up. A perusal of these papers shows how much we owe to Professor Munk and his insistence on the afferent basis of cortical disturbances.

Symptoms and their Interpretation. By James Mackenzie, M.D., M.R.C.P. Pp. 297. London: Shaw and Sons, 1909.

In this book, devoted mainly to a formal statement of his views on pain of visceral origin, the author brings his great practical experience of disease to bear on symptoms arising from reflex irritation of the nervous system. First he deals with the nature of pain, and insists that the viscera are completely insensitive. The causes of referred pain are discussed, and a chapter is devoted to increased sensibility of the external body-wall which so frequently accompanies it. The author insists on the importance of recognising the contraction of muscles evoked reflexly by visceral pain, so often misinterpreted by physicians. He then passes to a detailed consideration of the pain produced by the affection of the several organs based in every case on his own observations. Finally, the author sums up in a general estimation of the value of symptoms. There is a full index, which greatly facilitates reference to the many symptoms dealt with in this work.

A Text-book of Experimental Psychology. By CHARLES S. MYERS, Lecturer in the University of Cambridge. With 66 figures and diagrams, pp. 432. London: Edwin Arnold, 1909.

This is a most valuable handbook, and worthily supplies a long-felt want. In each chapter the student is given a short and readable account of some psychological question from the experimental standpoint. Every chapter has an appendix, in which the technical details of the elucidatory experiments are clearly described. The book begins with cutaneous sensations, and passes on through the special senses to the consideration of reaction-time, memory, local signature, identity and difference. Time and rhythm, attention and feeling, are also dealt with. The author is to be congratulated on the clearness and brevity of his explanations, in spite of the extensive ground covered by this work.

Lezioni di Anatomia Clinica dei Centri Nervosi. Per G. MINGAZZINI. With many illustrations, pp. 638. Torino: Unione Tipografico-editrice torinese, 1908.

This book consists of a series of lectures on the anatomy and functions of the central nervous system. In each chapter a single theme is dealt with and the anatomy and the functions of the part described. Thus three chapters are devoted to the spinal cord, eleven to the mid-brain and cerebellum, and four to the cerebrum. The author deals with the aphasia, alexia, and agraphia, and a lecture is devoted to the relation of speech and thought. The book is profusely illustrated with diagrams.

The Medical Annual for 1909. Pp. 744. With many illustrations. Bristol: John Wright; and London: Simpkin, Marshall and Co., 1909.

This volume is in every way up to the standard of its predecessors. Few fresh methods of treatment have been introduced into neurological practice during he last year, but the alcohol treatment of neuralgia receives full notice, and there is a good article on intracranial surgery. The illustrations are remarkably good.

Epilepsia. Revue internationale trimestrielle, consacrée à l'étude au point de vue pathologique, thérapeutique, social et juridique de l'Epilepsie et des Maladies nerveuses du même ordre. Sous le patronage de MM. W. BECHTEREW; O. BINSWANGER; J. HUGHLINGS JACKSON; L. LUCIANI; H. OBERSTEINER; F. RAYMOND; avec une Comité de la Rédaction. 1e Année, 2me Fasc. Avril—Juin, 1909. Amsterdam: Scheltema and Holkema.

This number contains papers by Binswanger, Allan McDougall, Donath, Muskens and Apelt. To each paper is appended a short summary in a second language.

Die Erkennung des Schwachsinns im Kindesalter. Von Professor Dr. TH. ZIEHEN. S. 32. Berlin: Karger, 1909.

This is an address to teachers and parents on the signs of mental defect in children. In simple language the abnormalities of the skull, the skeleton, and the general bodily development are shortly described, and the author then passes on to want of co-ordination and other similar defects. He then treats of mental defects which he groups under the heading of memory, comprehension, and judgment. This pamphlet would form an excellent guide to anyone anxious to put before teachers the simple signs of defective mental development.

Grundriss der psychiatrischen Diagnostik. Von Professor Dr. RAECKE. Mit 11 Textfiguren. S. 146. Berlin: Hirschwald, 1908.

This little book, which can be carried in the pocket, gives the various methods of examining the insane. It is adapted for German patients, but some similar pocket-book of methods is sadly wanting in English, and might perhaps improve the note-taking in our asylums.

Neurological and Mental Diagnosis: a Manual of Methods. By L. PIERCE CLARK, M.D., and A. ROSS DIEFENDORF, M.D. Pp. 188. New York: The Macmillan Co., 1908.

In the first half of this book the methods of neurological examination are described in order. It contains nothing new, and some of the methods advised are unnecessarily slipshod. The second half is devoted to the methods of examination in mental disease, but the general scheme is too short even for a student. It is difficult to see how the glossary at the end could be of use to any but an asylum attendant.

Nervous and Mental Diseases for Students and Practitioners. By Charles S. Potts, M.D. Second edition revised and enlarged. Illustrated with 133 engravings and 9 plates, pp. 570. London: Kimpton, 1908.

This is a small text-book on conventional lines. The symptoms which may occur in the course of any one disease are thrown together in a form so compact that the student can obtain no picture of the disease.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.

[NOVEMBER, 1909.]

BRAIN.

PART III., 1909.

Original Articles and Clinical Cases.

ON THE CERVICAL SPINO-BULBAR AND SPINO-CEREBELLAR TRACTS AND ON THE QUESTION OF TOPOGRAPHICAL REPRESENTATION IN THE CEREBELLUM

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AND
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INTRODUCTION.

THE object of the following investigation was to establish, if possible, what tracts ascend from the cervical region of the spinal cord to the bulb and to the cerebellum, for it is curious that hiatuses yet exist in our precise knowledge of the tracts which pass from the spinal cord to the cerebellum, one of the most notable being the absence of investigation into the relation of the spinal innervation of the fore-limb to the cerebellum. Indeed, as regards the spinal cord itself, practically the only attempt to compare the proportional representation of the fore- and hind-limb respectively in the spino-cerebellar paths is contained in the paper by Collier and Buzzard (*vide* Table A, p. 242). In the present work the examination of our material has also afforded an opportunity of considering how far anatomical research supports the views on the localization of function in the cerebellum which have been especially advanced by van Rijnberk on the anatomical ideas of Bolk.

As regards the first of these questions, while the source of the direct tractus spino-cerebellaris dorsalis is fairly well recognized and its destination partly ascertained, the origin of the tractus spino-cerebellaris ventralis, Gowers' tract, is almost as unknown to-day as when Edinger said of it in 1904 "der Ursprungsort ist noch nicht bekannt."

Its terminal distribution also to the vermis cerebelli, first ascertained by Mott, has since been variously described and requires fresh determination. Further, the origin and destination of the marginal fibres between these two great tracts and which compose the dorso-ventral tract of Pellizzi have received little attention, except from that author.

Our anatomical results will be classified according to each of these tracts in their dorsal and ventral order, but chiefly according to the metameric level of the cord from which the fibres arise. For this purpose, we may draw attention to the arrangement of the motor functions of the spinal cord which are in part subserved by the spino-cerebellar arcs of which Flechsig's and Gowers' tracts constitute the afferent side.

Arrangement of the Motor Representation of the Trunk and Limbs in the Spinal Cord.

Though the spinal cord must be regarded as a segmental mechanism, it is also the seat of special groupings of nerve centres (Goltz, Sherrington, Lapinsky) innervating the musculature for movements of the head and neck, of the fore-limb, the trunk and the hind-limb. It is the association of such compound central mechanisms with the cerebellum that is subserved by the spino-cerebellar paths, and therefore it appears to us that the afferent impressions to the small brain may be considered as originating in one or other of the following regions:

Region I ...	1 C. to 4 C.	Rotation of head and neck	Flexion, extension, lateral adduction of head and neck.
Region II ...	5 C. to 1 D. ...	Fore limb ...	
Region III ...	2 D. to 2 L. ...	Trunk ...	Flexion, extension, lateral adduction of trunk.
Region IV ...	3 L. to 2 S. ...	Hind limb	

Hence each of the fundamental subdivisions or parts of the body may schematically be regarded as represented in one of four regions of the cord (Region I., II., III., IV.), which may conveniently be termed the head and neck region, the fore-limb or arm region, the trunk region, and the hind-limb region respectively.

A crude classification of this sort also has the advantage of conveying reference to the physiological relationships of these parts of the body.

HISTORICAL SURVEY.

The general literature of the spino-cerebellar tracts is well known, but it appeared to us, in endeavouring to map out the destination of the tract-fibres, that it would be useful to tabulate the knowledge already gained as follows:—

Table A.—Historical Table of Gowers' Tract (Tractus spino-cerebellaris ventralis).

Author	Reference	Nature of Research	Abstract
Bastian .	*Med. Chir. Trans.*, 1867, p. 499	Case of paraplegia	The tract consists of afferent fibres and is part of the dorsal or direct cerebellar tract.
Gowers . .	"Diagnosis of diseases of the spinal cord," 1879	Cases of paraplegia	Probably conducts painful sensations from the opposite side of the body; its component fibres probably arise from cells in the posterior commissure.
Singer ...	*Sitzungsb. d. K.K. Akad. d. Wissensch. Wien*, 1881, Bd. lxxxiv	Experimental degenerations	Considered tractus ventralis a spino-cerebellar one.
Testut . .	Traité d'anatomie humaine, vol. ii., p. 352	Anatomical evidence	Divides cerebellar tract into ventral and dorsal portions and states that their fibres thus directly represent long anastomoses between the superior vermis of the cerebellum and Clarke's column.
Löwenthal ...	*Revue Med. de la Suisse Romande*, 1885, p. 511.	Experimental degeneration	Lesion between fifth and sixth pairs of cervical nerves. Whole of left lateral column destroyed. Ventral portion of cerebellar tract is traced up to where it turns towards the external periphery of the superior cerebellar peduncle, and then runs backwards describing outside the peduncle a prolonged half-turn and eventually appears at the dorso-internal side of the superior cerebellar peduncle at the spot where the peduncle reaches the medullary velum of the cerebellum.

Author	Reference	Nature of Research	Abstract
Singer .	"Ueber die Veranderungen im Ruckenmark nach zeitweiler Verschliessung der Bauch Aorta," *Sitzungsb. d. K. K. Akad. d. Wissensch. Wien*, 3 Abth., 1887, 95-96	Compression of abdominal aorta Experimental degeneration	Origin of tract is in cells of anterior cornu in the lumbar enlargement.
Auerbach	"Zur Anatomie der aufsteigend degeneriren den Systeme des Ruckenmarks," *Anat. Anzeiger*, 1890, No. 3 u. 4. Also "Zur Anatomie der Vorderseiten-strangreste," *Arch. f. Pathol. Anat. u. Physiol.* v. Virchow, Bd. 121, 1890, S. 199	Experimental degeneration	Posterior part of half spinal cord destroyed for some length. (Posterior white column, posterior horn of grey matter and posterior part of lateral column.) Degenerations in ventro-lateral cerebellar tract up to ventral part of superior vermis of cerebellum The number of degenerated fibres in anterior and antero-lateral column decreases from below upwards.
Edinger ...	"Einiges vom Verlauf der Gefuhlsbahnen im Centralen Nervensystem," *Deutsch. med. Wochenschr.*, 1890, S. 421	Anatomical work on spinal cords of fishes, reptiles and amphibia	Antero-lateral tract is probably connected with transmission of painful sensations from opposite side, but fibres come from cells of posterior horn and decussate in anterior commissure. Section of posterior roots causes no degeneration in the antero-lateral column.
Bechterew ...	*Neurol. Centralbl.*, 1890, S. 354	Myelinisation method	Gowers' tract undergoes development at a different period from the rest of the lateral column.
Mott	"Ascending Degeneration resulting from Lesions of the Spinal Cord in Monkeys," *Brain*, vol. xv, 1892, p 215	Experimental degeneration	Antero-lateral tract divided either on one side or both sides. Ventral cerebellar tract may be completely divided in monkeys apparently without producing analgesia. Gowers' tract traced to the ventral part of the superior vermis by way of the superior cerebellar peduncle forming in its course a remarkable loop over the fifth nerve. Scattered fibres belonging to the fillet system can be traced as far as the anterior corpus quadrigeminum of the same side.

Author	Reference	Nature of Research	Abstract
Grünbaum .	*Journ of Physiol.*, 1894, vol. xvi, p 368	Longitudinal division of the spinal cord	Found an ascending degeneration at the anterior border of the cerebellar tract.
		Injury to anterior cornua. Degeneration evidence	In sections above the lesion degeneration is found on both sides of the cord in the cerebellar and antero-lateral tracts.
Mott	"Experimental Enquiry upon the Afferent Tracts of the Central Nervous System of the Monkey, *Brain*, vol. xviii, 1895, p. 1	Experimental degeneration after longitudinal division of spinal cord	Unilateral section of posterior roots. Degenerated fibres sometimes found in opposite antero-lateral column. Assumes (1) These fibres come from cells of grey matter of the same side as the lesion and cross over in the anterior commissure, or (2) were damaged fibres due to vascular changes in the grey matter, the result of operation.
Pellizzi ...	"Sur les dégénérescences secondaires dans le systême nerveux central a la suite de lésions de la moelle, et de la section de racines spinales," *Arch Ital. de Biol.*, t. xxiv, 1895, p 89	Experimental degeneration	Ventral cerebellar tract follows course of superior cerebellar peduncle, spreads out feather-wise (*en panache*) and crossing with the fibres of the opposite side radiates into the white substance of the cerebellar folia of the anterior and inferior part of the superior vermis Before crossing in the middle line a large bundle goes upwards to the superior portion of the superior vermis. He suggests that these fibres cross successively higher up. No fibres seen going to cerebellar nuclei. After section of posterior roots (lumbo-sacral and cervical) he observed degenerations in the cerebellar tracts!
J Risien Russell	*Brain*, 1898, vol. xxi, p. 145	Case of tumour of spinal cord. Degeneration evidence	Gowers' tract begins at the level of the third lumbar.
Schäfer ..	*Journ. of Physiol.*, 1899, vol. xxiv, p. xxiii	Chromatolysis evidence	States some fibres of Gowers' tract arise from cells of Clarke's column.
O. Kohnstamm	*Neurol Centralbl*, March,1900, S. 242	Degeneration evidence	The lesion destroyed the posterior quadrant of the cord, including the posterior horn In Gowers' tract the more proximally arising fibres lie median to fibres arising further back.
Lubouschine ...	*Le Nevraxe*, vol. iii, 1901-2, p. 123.	Degeneration evidence	Diffuse injection lesion of posterior cornu. Concluded that the fibres of Gowers' tract arise from the posterior cornu of both sides.

Author	Reference	Nature of Research	Abstract
Collier and F. Buzzard	*Brain*, 1903, vol. xxvi, p. 559	Degeneration evidence from clinical cases	First discussed fully the relative regional origin of the spino-cerebellar tracts. Probably less than one-half of the tractus spino-cerebellaris dorsalis is cervical and thoracic in origin. Found that a mesial bundle of the tractus spino-cerebellaris dorsalis gave collaterals to the nucleus dentatus, and inferior vermis (lobes ?). The lateral main bundle also gave collaterals to the nucleus dentatus. Degenerate fibres traceable to all parts of the cerebellar cortex especially the lower part of the lateral lobe. Regard spino-vestibular fibres as of lumbo-sacral origin only. Also that Gowers' tract sends fibres to both lateral lobes and to inferior vermis, also to the dentate nucleus as well as the nucleus fastigii.
Quensel and Kohnstamm	*Neurol. Centralbl.*, 1907, S. 1138	Degeneration evidence	Regard the whole Gowers' system as arising from the posterior cornual cells of the contra-lateral side.
Schäfer and Symington	*Quain's Anat.*, 11th Ed., 1908, vol. iii, pt. 1, pp. 85, 97, &c.	Epitome of all evidence	"The dorsal and ventral spino-cerebellar tracts perhaps have a common origin, possibly from the cells of Clark's column, but also from other cells in the grey matter of the same and perhaps of the opposite side."

Table B.—Historical Table of Flechsig's Tract (Tractus spino-cerebellaris dorsalis).

Flechsig ...	"Leitungsbahnen im Gehirn u. Rückenmark," Leipzig, 1876	Myelinisation evidence	The tract is derived from cells of Clarke's column. III lumbar to II thoracic segments inclusive in the dog.
Lowenthal	*Revue Med. de la Suisse Romande*, 1885, p, 511	Lesion between fifth and sixth pairs of cervical nerves. Whole of left lateral column destroyed	The dorsal portion of the direct cerebellar tract terminates in the restiform body. He gives reasons for connecting its fibres with the superior vermis of the cerebellum.
Auerbach ...	*Vide* reference in Table of Gowers' tract, 1890	—	Traces dorso-lateral cerebellar tract up to dorsal portion of superior vermis.
Mott	*Brain*, 1890, vol. xiii, p. 433	Degeneration evidence	The tract takes origin in the cells of Clarke's column.
	Brain, 1892, vol. xv p. 215	Antero-lateral tract divided either on one or both sides	The dorsal cerebellar tract connects cells of Clarke's column with the dorsal portion of the superior vermis.

Author	Reference	Nature of Research	Abstract
Pellizzi ..	*Vide* reference in Table A, 1895	Degeneration evidence	Tract radiates towards the superior anterior and inferior part of the superior vermis of the opposite side decussating in the middle line with the restiform fibres of the other side. States that Mott's conclusions with regard to termination of tracts are incorrect. The majority of the cerebellar tract fibres take origin from the cells of Clarke's column; where these are not present, from groups of cells which represent them.
H. T. Patrick	*Journ. of Nerv. and Mental Dis.*, xxiii, 1896, p. 85.	Degeneration evidence	Confirms Mott.
Van Gehuchten	*Journ. de Neurol.*, Bruxelles, 1898	Chromatolysis evidence	Confirms Mott with regard to origin of tract from Clarke's column.
Schäfer ...	*Journ. of Physiol.*, xxiv, March 18, 1899, p. xxii and xxxii	Degeneration evidence	Confirms Mott.
Warrington ..	*Journ. of Physiol.*, xxiv, 1899, p. 476	Chromatolysis evidence	States also that cells are distinguished by chromatolysis and early atrophy with absence of tendency to repair characteristic of other cells.
Sherrington & Laslett	*Journ. of Physiol.*, March, 1903, p. 58	Method of "successive degeneration"	(1) Confirm Mott and Warrington. (2) Further state that the tract is stratified in the following order: Outermost—Fibres from segments furthest backward in the cord. Next — Fibres from the lowest thoracic segments. Deep to these —Fibres from thoracic segments further forwards. Innermost — Fibres from topmost thoracic and lowest cervical segments. (3) Demonstrate — That some of the fibres of the tract are long internuncial spinal fibres.
Edinger .	"Text-book," 1890, p. 327	Anatomical investigation, &c.	The tract comes from vesicular column of Clarke, is a secondary continuation of posterior root-fibres, constitutes the main part of the spinal connection, and curving mesially enclosing the corpus dentatum, passes to the cortex of the superior vermiform process.
R. Bing ..	"Experimentelles zur Physiologie der Tractus spino-cerebellaris," *Arch. f. Physiol.*, 1906, S. 250	Physiological and degeneration evidence	After section of direct cerebellar tract in dogs found that the animals stand with abducted legs and are atactic. States that lesion of cerebellar tracts involves two primary considerations: (1) Lesion of special method of control. (2) Lowering of muscle tonus.

Author	Reference	Nature of Research	Abstract
Otto Marburg	"Die physiologische Function der Kleinhirnseitenstrangbahn," *Arch. f. Anat u. Physiol.*, 1904, S. 457	Degeneration evidence	Shows that tractus spino-cerebellaris dorsalis has the most important share in the regulation of the "principal" movements.
Rothmann ...	*Neurol. Centralbl.*, 1907, S. 968	Degeneration evidence	Nucleus lateralis inferior, though receiving fibres from the tractus spino-cerebellaris dorsalis, receives none from the tractus spino-cerebellaris ventralis.
Munzer and Wiener	*Arch. f. experimentelle Pathol.*, 1895, Bd. 35, S. 113	Degeneration evidence	

Table C.—Other Spino-Cerebellar Tracts Described.

G. B. Pellizzi	*Vide* Table A ...	*Vide* Table A ...	*The median cerebellar tract.* To dorsal and ventral cerebellar tracts a median tract must be added equally constant although smaller. Lies between dorsal and ventral cerebellar tracts in cord and medulla, is composed of large fibres. At the emergence of N. VII its fibres tend dorsally. Then the fibre go further dorsally inwards and lie along the internal border of the ventral extremity of the ascending root of V. It thus comes into contiguity with the ventral tract, but the fibres of the two tracts are always distinct from one another. The fibres are abundant in the caudal half of the middle peduncle and lie in the internal part of its radiations. They go towards the antero-superior part of the superior vermis.
Rothmann ..	*Arch f Anat. u. Physiol.*, Physiologische Abtheilung, 1899, S. 120	Degeneration evidence.	*A sacro-lumbar lateral cerebellar tract* is present in dogs and lies above ventrally to dorsal cerebello-spinal tract. Many of its fibres end in the upper dorsal and cervical cord, but others pass into the medulla as a lateral cerebellar tract. Considered (p. 46) a larger part of Gowers' tract crossed the middle line of the cerebellum (1 experiment).
	Neurol. Centralbl, Nos. 1 and 2, 1900, S. 16	By embolus of cord with semen Lycopodii excluded function of conus terminalis and lumbo-sacral cord. Thus obtained degeneration of tract in posterior part of the lateral column	

From this survey of what has already been accomplished it will be seen that nothing has been yet done definitely to ascertain the relation of the spinal centres subserving the fore-limb and head and neck musculature to the cerebellar mechanism although the association of these parts is very intimate owing to the requirements of equilibration.

It appeared to us desirable to take advantage of the increased knowledge of the cell grouping in the spinal cord gained by the researches of Bruce, Parhon, and others, knowledge which has been lately supplemented by the researches of Harvey Pirie, and especially of Jacobson on the middle cell groups of the spinal cord, particularly those which occupy (1) a position in the cervical cord homologous with the cells of Clark's column, (2) the middle region of the grey cornu. From Jacobson's work, it seemed clear that although throughout the cervical spinal cord large and characteristic cells are occasionally seen in the situation of Clarke's column, the spino-cerebellar paths in the cervical regions must arise from the middle cells of the cornu.

The present research was designed and begun before the more recent normal anatomical work just referred to was published, but we had decided on grounds of homology to attempt to destroy the middle region of the cornu and therefore the "middle cells" by means of a small longitudinal lesion.

It seemed that the cells and origin of the tractus spino-cerebellaris ventralis might be regarded as lying approximately in front of a line passing through the posterior or dorsal limit of the central canal and not in the posterior cornu. It was therefore necessary to employ some means of causing the destruction of very limited areas within the boundaries of the cornu.

At the same time, in order to study separately the distribution of the cervical (*i.e.*, head and neck and fore-limb) spino-cerebellar fibres from those coming from the other regions of the cord and belonging to the trunk and hind-limb, the antero-lateral column had to be carefully preserved from injury. The injection method employed by Münzer and Wiener and subsequently by Lubouschine causes such widespread damage and is limited with so much difficulty that we did not employ it and resorted to electrolysis.

Operative Procedure and Material.

The operative procedures adopted were carried out by one of us (V. H.) as follows:

The spinal cord was exposed in the anæsthetised animal by removing the laminæ of two to three vertebræ; a minute lesion was made in the spinal cord parallel to the central canal by means of a short electrolytic needle fitted with a suitable handle and bent at a right angle. The anatomical direction given to the needle was penetration (vertically to the cord) of the posterior root zone and then headwards paralled to the central canal. The needle was made an anode, the kathode being placed on the neck muscles exposed in the wound, and electrolysis (anodal) was then performed. This in the majority of the experiments comprised the passage of a current of 1 milliampere for thirty seconds. For a larger lesion 2 ma. for one minute was employed.

The animal was kept for three weeks and then killed under chloroform after the reflex arcs and the conductivity of the cord has been tested by faradic excitation. The resultant degeneration in the brain and spinal cord was studied by the Marchi method. Excluding the cases in which the experiment failed for different reasons, we obtained complete results from one dog, two cats and eight monkeys (six *Macacus rhesus*, one *M. cynomolgus*, one *M. sinicus*).

In addition we have employed a number of sectional series of unpublished exclusion experiments in cats and monkeys made during the last eight years by one of us (V. H.), including direct and very restricted lesions of the ascending spino-cerebellar tracts and at various levels in the bulbo-spinal axis.

RESULTS.

I. — *Course of Spino-cerebellar Fibres in the Various Regions of the Spinal Cord.*

REGION I.

I.—*Contralateral Fibres.*

Taking the regions of the cord enumerated on p. 238, and beginning with Region I, we find that the contra-lateral fibres from cells in the dorso-medial area (Pirie's para-central and Clarke's column areas) of the grey cornu cross in the anterior commissure and through the anterior cornu to gain the contra-lateral ground bundle in which they ascend lying dorsally to the ventral spino-cerebellar tract formed by the fibres from the rest of the spinal cord.

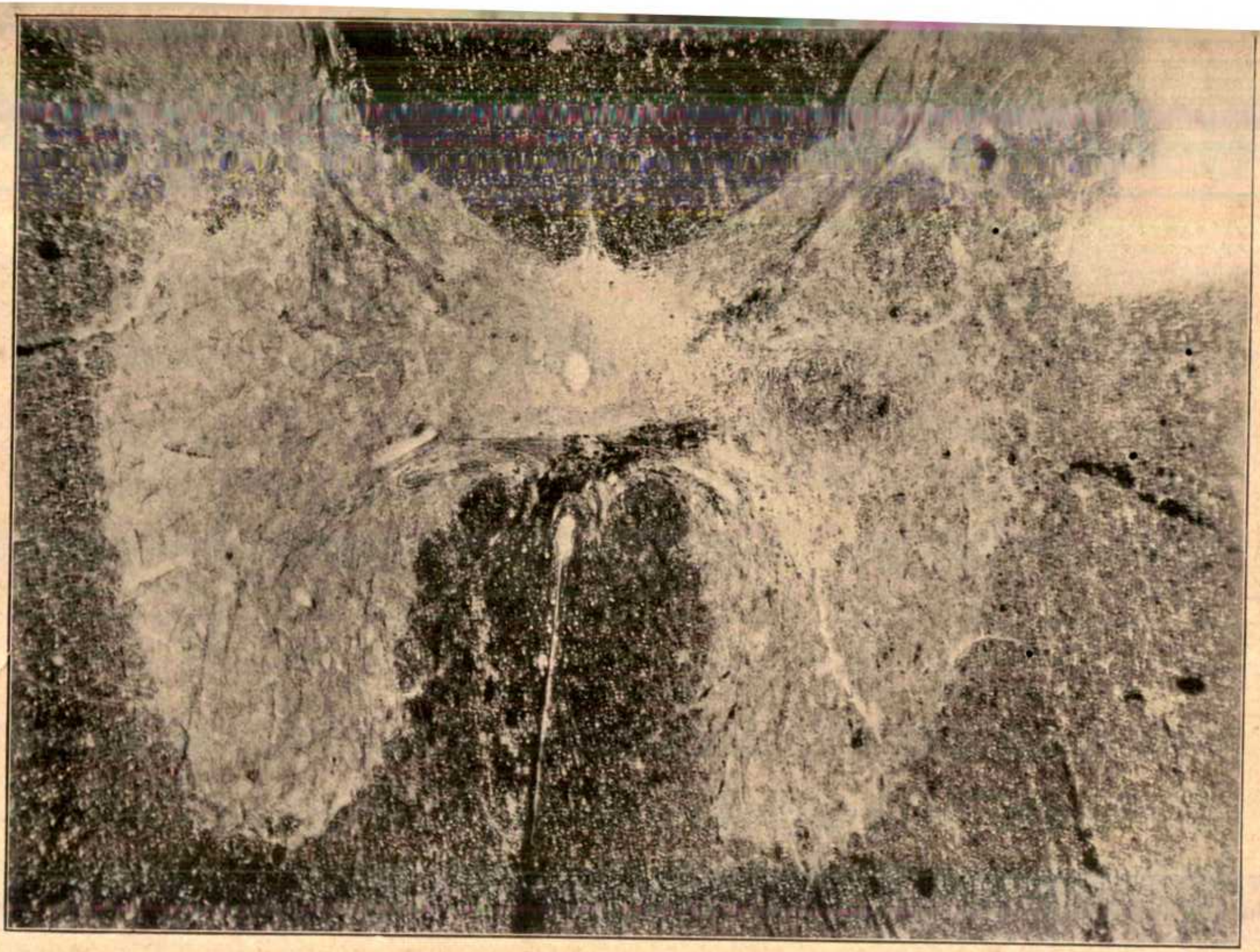

FIG. 1.

Section of the spinal cord (*Macacus rhesus* 32) at the level of the second cervical root, showing the nature and situation of the lesions employed in the present research as well as the main paths of the fibres leading therefrom.

These large fibres ascend in the pars reticularis lateralis medullæ and in *Macacus rhesus* terminate in the lobus pyramidalis of the cerebellum.

(2) The ipsilateral fibres consist of two groups, one springing from a position mesial to the intermedio-lateral area (Pirie's anterior central, central, and post-central areas) of the cornu and entering the lateral column to reach the nucleus lateralis or passing up behind the fastigio-brachial bundle near the lobus centralis and anterior part of the lobus pyramidalis. Summing up these results, therefore, it is clear that the spino-cerebellar fibres springing from the upper cervical region (head and neck movements) pass to all portions of the vermis except the most dorsal and most ventral, the parts omitted being the two anterior lobuli of the lobus posterior (Elliott Smith) and the lingula, nodulus, and uvula.

Further, these fibres, like those from lower spinal levels, give off in the medulla collaterals to the nucleus vestibularis (Deiter's), the nucleus formationis reticularis, and the nucleus lateralis.

REGION II.

The fibres from this area of the cord are important since they constitute the spino-cerebellar tracts for the upper limb. Some of them pass to the tractus dorsalis; we may therefore commence with the the ipsilateral class.

(1) *Ipsilateral Fibres.*

Dorsal Fibres.—A few fibres pass towards the tractus spino-cerebellaris dorsalis forming the most internal group and occupying longitudinally a length of three segments in attaining their position in the area of the tract.

Dorso-ventral.—Fibres of varying diameter from the intermedio-lateral region of the cornu pass up in the lateral column at a distance from the peripheral margin of the cord to the nucleus reticularis medullæ and to the nucleus lateralis medullæ.

Ventral Fibres.—The ventrally running fibres tend to form Gowers' tract in the usual way, but they remain in the ground-bundle area of the lateral column and do not reach the surface of the cord. The most internal pass in the medulla close to the outer margin of the spinal fifth and ascend to the cerebellum lying between the fibres of Gowers' (dorso-lumbar) tract and the fastigio-brachial bundle (fasciculus fastigio-bulbaris).

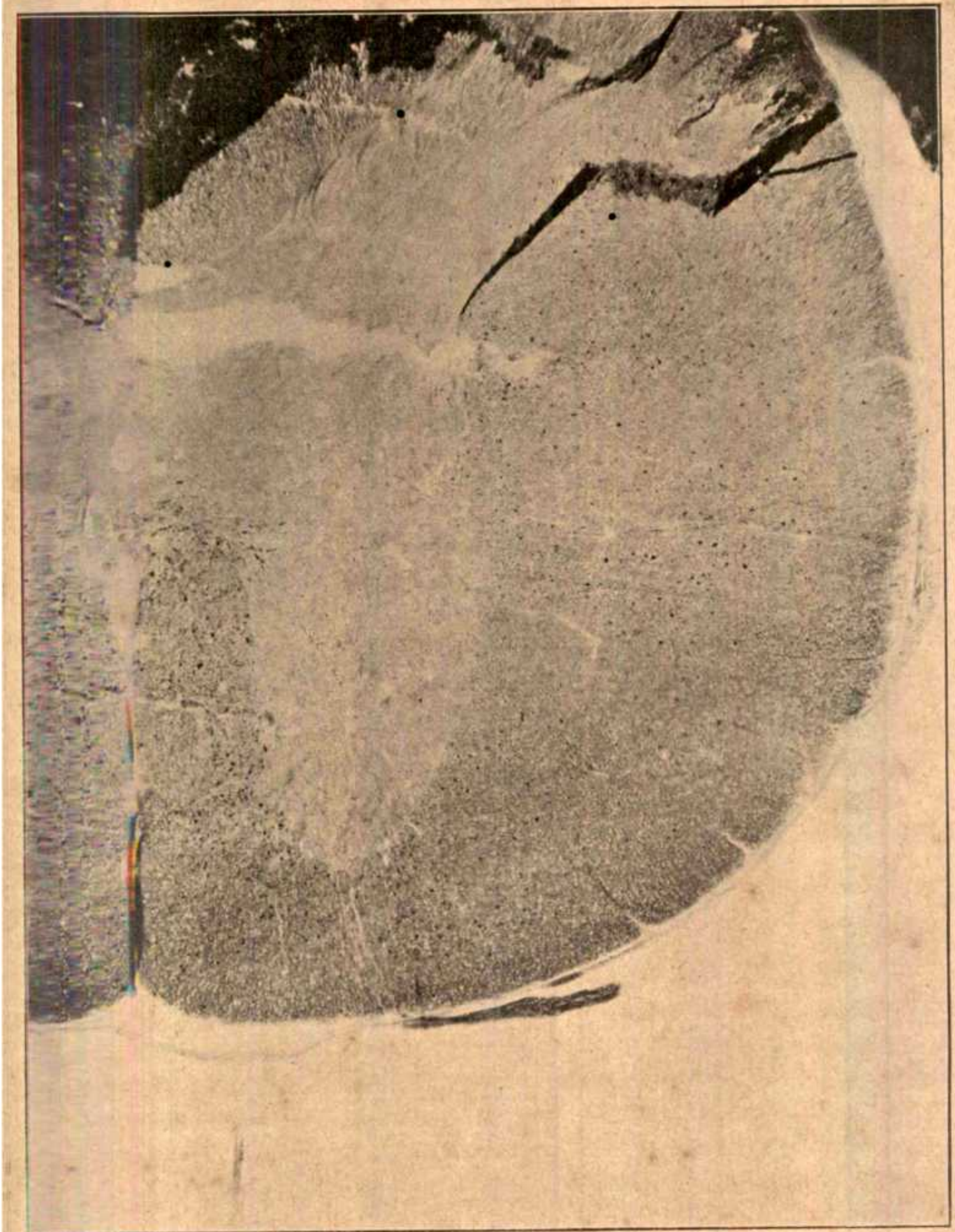

FIG. 2.

Section of the spinal cord (*Macacus rhesus* 36) at the level of the first cervical root, showing the ipsilateral fibres in the lateral column.

Thus in confirmation of Kohnstamm's observation we find that the spino-cerebellar fibres for the fore-limb occupy definitely a position dorsal, i.e., posterior to those for the trunk and lower limb but in their distribution and termination in the lobus centralis they are scattered over the whole area, and are not aggregated into any special folium. This general arrangement of the spino-cerebellar fibres in laminæ is the same as that originally pointed out by Sherrington and Laslett.

(2) *Contra-lateral Fibres.*

These are of very varying diameter and on the average smaller than those of the ipsilateral side. They run up in the ground-bundle, gradually passing outwards, and only arriving at their place in the main path after passing through about five segments of the cord. In the formatio reticularis they lie ventrally and internally to the trigeminal spinal root.

These fibres are internal to, and do not appear to send collaterals into the nucleus lateralis of the medulla. As they ascend in the formatio reticularis they gradually diminish in number.

REGIONS III AND IV.

As regards these regions of the cord we have made no special experiments on Region III., and as regards Region IV., we found in control experiments that the course of the fibres in the spinal cord was as described in the text-books, and needs no repetition. The termination of the various groups of fibres in the cerebellum we will now discuss.

II.—*The Terminal Distribution in the Cerebellum of the Spino-cerebellar Tracts.*

Pellizzi, in adversely criticising the original statements of Mott relative to the termination of the spino-cerebellar paths in the cerebellar cortex appears to have fallen into the error of basing his opinions on extensive lesions of the cord only. Our experience from restricted lesions, on the contrary, confirms Mott's views, and we will now state in summarised form the results in detail.

REGION I.

The spino-cerebellar fibres of this region, which are in the main furnished through the tractus spino-cerebellaris ventralis, are distributed to the lobus centralis and culmen. None pass to the lingula.

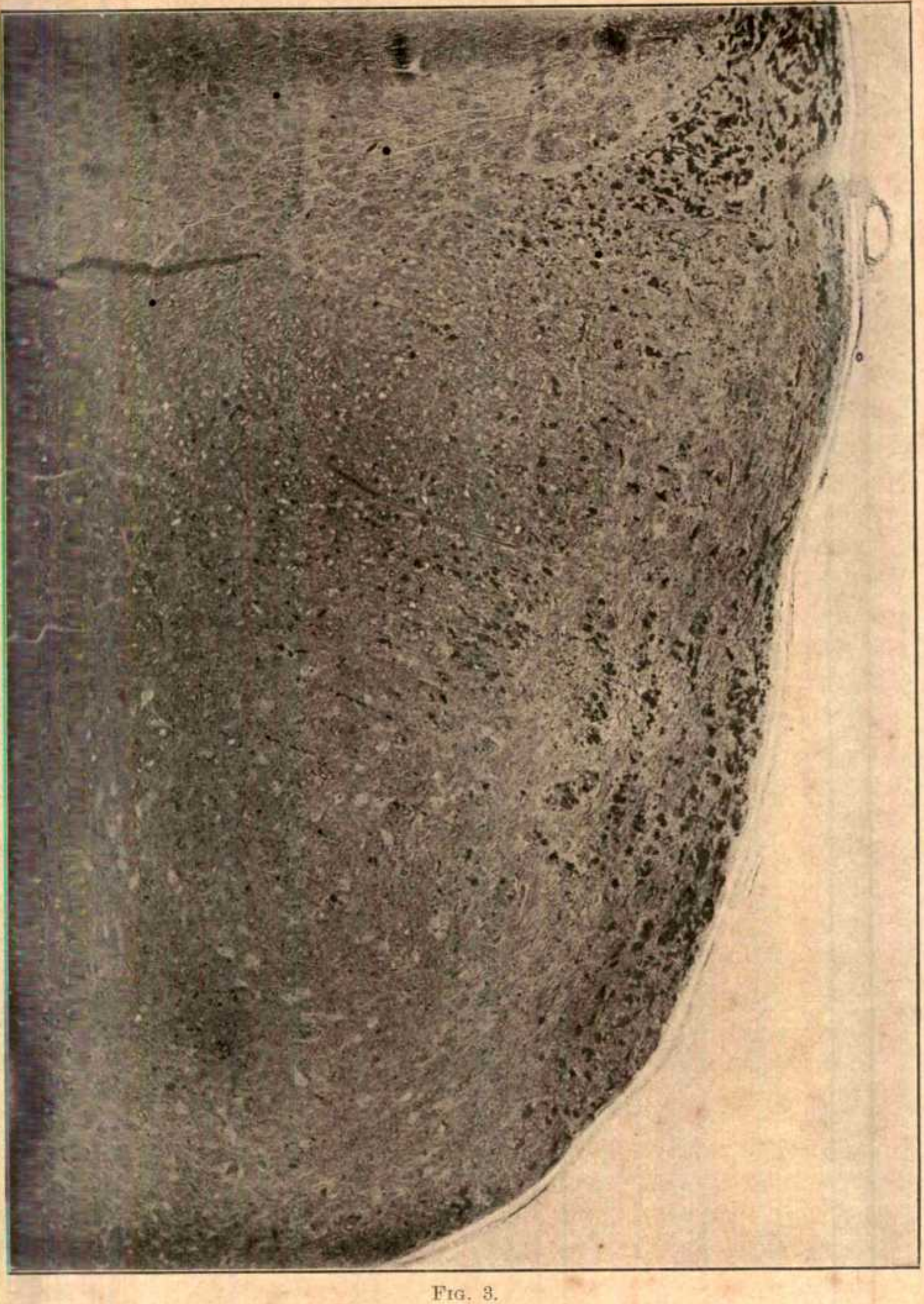

FIG. 3.

Transverse section through the bulb at the centre of the nucleus lateralis from an extensive (5 segments) lesion of the mid-cornual region in the cat, showing fine collateral fibres degenerated among the cells of the nucleus lateralis.

A few are distributed to the uppermost folia of the lobus pyramidalis *i.e.*, through the tractus spino-cerebellaris dorsalis).

Region II.

The fibres of this region, *i.e.*, arising from the fifth cervical to the first dorsal spinal segments, end in the cortex of the cerebellum as follows the lobus centralis, the ventral half of the culmen, and the ventral half of the lobus pyramidalis.

In full agreement with Mott's results we have found that the fibres of Gowers' tract (purely isolated) are practically not distributed to any part of the cerebellar cortex behind the plane of the anterior cerebellar commissure, except the collaterals which are distributed to the nucleus fastigii of the same side. Further that the distribution to the cortex is by medium-sized collaterals[1] springing from the main fibres as they cross in the anterior commissure.

These collaterals enter all the folia of the lobus centralis and ventral part of the culmen. A striking illustration of the fact that the fibres of the tractus ventralis from the hind-limb region, Region IV., of the spinal cord are distributed to exactly the same area of the cerebellar cortex as those from the fore-limb region is shown by two experiments (62 and 80, 1903, V. H.), in which extremely limited lesions of Gowers' tract were made at the first lumbar level and the third and fourth cervical level respectively. In both animals the degenerated fibres were precisely traced to the same point in the cerebellum, viz., the lobus centralis and to the ipsilateral nucleus fastigii, no fibre passing to the cortex cerebelli anywhere caudal to the posterior plane of the commissura anterior.

Region III.

We have not made any experiments on this region.

Region IV.

The termination of the spino-cerebellar fibres in the cerebellum from the caudal limb region may be limited to the discussion of the tractus spino-cerebellaris dorsalis.

According to our observations the whole tractus spino-cerebellaris dorsalis ends by freely branching fibres in the lobus centralis, the culmen, the lobus posterior, and the lobus pyramidalis on both sides of the median plane, the ipsilateral fibres being to the contralateral as 2 to 1.

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[1] These collaterals subdivide very rapidly in penetrating the substantia alba of the folia.

Mott's original description is in general principle thus fully established, and it is clear that whereas the tractus ventralis is distributed principally to the folia of the lobus centralis and to a much less extent the culmen and lips of the primary fissures the tractus dorsalis not only supplies the same cerebellar cortex, but extends backwards throughout the lobus pyramidalis. Thus, the area of distribution of the tractus ventralis is anterior or headward compared to that of the tractus dorsalis.

We cannot leave this most important question without drawing attention to a reversal of the facts bearing upon it which has curiously received support so that even in the last edition of Quain's "Anatomy," vol. iii., pt. 1, 1908, p. 199, the statement is unfortunately made that "The fibres from Gowers' bundle are distributed mainly to the caudal half of the vermis on both sides."

III.—*The Connection between the Spinal Nuclei and Cerebellar Nuclei.*

(1) *Via the Tractus Spino-cerebellaris Ventralis.*

We have already discussed the supply of the tractus spino-cerebellaris ventralis to the cortex of the cerebellum. We must now discuss the precise nature of the important connection between this tract and the nucleus fastigii (roof nucleus), a connection the existence of which has been questioned. This association, which was described in man by Collier and Buzzard, is provided by numerous fibres in the rhesus and in the carnivora (cat, dog).

As the fibres of the tract cross in the anterior commissure, they send stout collaterals back between the nuclei fastigii within which finer branches of these fibres ramify throughout the whole nucleus. The spino-nuclear connection is almost wholly unilateral, few fibres entering the contralateral nucleus.

(2) *Via the Tractus Spino-cerebellaris Dorsalis.*

The dorsal spino-cerebellar tract has, so far as we can see, no connection in the cat or rhesus with the nucleus dentatus, although the fibres curve round the nucleus to reach the cortex and although such communication was observed in man by Collier and Buzzard. Absence of such connection would be, of course, in harmony with the fact that the nucleus dentatus is in relation with the cortex of the lateral lobe and therefore not, like the vermis and nucleus fastigii, in direct association with any spinal path (for the cortical relations of the nuclei, see

Clarke and Horsley, *Brain*, 1905, p. 13), but the proximity of the tracts to the fleece of the nucleus suggests an intercommunication and the point is worthy of a special reinvestigation.

The distinction between the vermis and the lateral lobes generally recognised is thus intensified.

IV.—*On the Question of the Topographical Localization of the Limbs and Part of the Body in the Cerebellum.*

From the foregoing account of the distribution of the cervico-cerebellar fibres especially those of the ventral tract, it is quite clear that by their distribution over the whole area of the vermis, which is allotted to the spino-cerebellar system, each part of the spinal cord must, practically speaking, be represented in every unit of the cortex to which the spinal fibres run. From the point of view of afferent function, therefore, there cannot be said to exist any evidence of differentiation of the cerebellar cortex into localized receiving stations for the impressions (muscular, arthric, etc.), which ascend from the arm, trunk, or leg muscles, joints, etc., respectively.

It would appear, therefore, that the cerebellar cortex is a structure in which these muscular-sense impressions are associated together, or to use a more frequently employed expression, co-ordinated. The recent excitation and exclusion experiments by which it is believed that definite centres for the movements of different parts of the body have been demonstrated to exist in the cortex cerebelli, just as in the cortex cerebri, must, we think, be otherwise interpreted. Particularly is this the case, where exclusion experiments have been made by extirpation of parts of the cerebellar cortex, for there is no doubt that the error pointed out originally by Clarke and Horsley has not been sufficiently avoided, and that the phenomena noted have been due at least as much to lesions of the subjacent nuclei, the nucleus fastigii, dentatus, etc., and even of the nucleus vestibularis, as of the cortex cerebelli.

V.—*Symptoms Displayed by the Animals in whom Lesions of the Cervical Spino-cerebellar Tracts had been made.*

Our clinical observations on the experimented upon animals, though furnishing only few symptoms and those remarkably limited in degree owing to the very restricted character of the lesions, are, nevertheless, confirmatory of the results obtained by Marburg and by Bing.

The first notable point is the question how far interference with the spino-cerebellar tracts causes a loss of efferent (purposive) movement. No such loss was observed by us. The motor effects we observed were invariably transitory and attributable to the direct interference with the anterior cornu, or to the effects of the anæsthetic. As regards equilibration, however, and accuracy of movement in all the animals experimented upon there was to be seen ataxy and clumsiness of movement most noticeable in the upper limb. With the ataxy there was very frequently contracture and the arm of the same side was often in the sling position as described by Mott and Sherrington, after division of the (brachial) posterior roots.

On the Unilaterality of the Distribution of the Spino-cerebellar Tracts.

Several authors have noticed that the fibres of the tractus spinocerebellaris dorsalis are distributed, more or less, to both sides of the middle line of the vermis. The bilateral distribution, both spinocerebellar tracts, merits a quantitative consideration and the material at our disposal enables us to estimate this roughly. The tractus spino-cerebellaris dorsalis is bilaterally distributed to twice the extent of the tractus ventralis the figures being approximately

	Cerebellar distribution	
	Ipsilateral	Contralateral
Tractus dorsalis	2 ...	1
Tractus ventralis	4 ...	1

THE CONDITIONS OF FATIGUE IN THE NERVOUS SYSTEM.[1]

BY WILLIAM McDOUGALL, M.A., M.B.

OUR English word "fatigue" is a most comprehensive one. Wherever there is evidence that organic activity results in diminished capacity of the organism to carry on any of its functions, there we commonly recognize *fatigue*. Such evidence is of two kinds, objective and subjective. Of the subjective symptoms we must distinguish at least three classes:—

(1) Local sensations of fatigue, more especially in the muscles.

(2) The sense or feeling of *general tiredness*, of limpness and general incapacity for effort.

(3) The experience we call *sleepiness*.

The first of these I dismiss with a single observation You may get this sensation of local fatigue in the acutest form by holding out a limb —say, the arm—for a few minutes against the pull of gravity. After a few minutes you begin to experience in the muscles of the shoulder a disagreeable sensation, which grows rapidly more intense, until, after about five minutes, you feel that you can hardly support it. But, if you persevere, you find that there is no impossibility about doing so; the disagreeable or painful sensation grows but little more intense, if at all, after the first few minutes; you may continue to hold out your arm, and, after half an hour, there will be little more discomfort than after five minutes.

Put alongside that the fact that most good hypnotic subjects will, when in hypnosis, keep a limb held up against the force of gravity for long periods without showing any tendency to relax it, or any other symptom of fatigue.

Facts of this order seem to show clearly that such local fatigue-sensation is protective in function; that in sets it long before there is any question of exhaustion of, or serious injury to, the tissues concerned through over-action. They show that the organism has need of some protection against the possibility of self-injury through excess of activity.

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[1] The ideas set forth in this paper were embodied in an address to the Physiological Section of the British Association at the Dublin meeting in the year 1908. That address was printed in the *Reports* of the Association, but I have sought from the editor of this journal the opportunity of bringing my ideas more prominently before the medical world by publication in these pages.

And this conclusion may serve to direct our attention to the possibility of a similar function being performed by other manifestations of fatigue.

In connection with the two other classes of subjective symptoms—tiredness and sleepiness—I would draw attention to experiences of a kind that are familiar to all of us. I sit reading in the evening some difficult technical work, until, as midnight approaches, I feel thoroughly tired and sleepy, my eyes smart and will hardly keep open, my head is dull and heavy and aches a little, my whole body and my limbs feel heavy and incapable of alert movement. I then take up some thrilling story or other fascinating piece of literature; whereupon all my subjective symptoms of fatigue, both tiredness and sleepiness, disappear, and I read keenly for perhaps two hours; when I again begin to feel both tired and sleepy. Now suppose a continuation of the evening's history which is less familiar: some exciting event takes place just as I am about to go to bed—the house takes fire, or a burglar breaks in, or a friend comes in with important and interesting news that demands immediate action. Again all the symptoms of fatigue disappear, and I can perform another spell of work at high pressure, without subjective symptoms, and with little, if any, objective trace of fatigue, *i.e.*, without easily appreciable diminution of efficiency.

Facts of this order show clearly, I think, that what we commonly call fatigue, with all its diverse symptoms, objective and subjective, is no entity, that it is not due to any one kind of change in the organism; and they show that fatigue is never *absolute*, but is always *relative*. That is to say, the symptoms of fatigue are always the expression of the relation between at least two things—between, on the one hand, the work to be done, and, on the other hand, the amount of energy available at the moment for doing the work; or, in more technical language, the symptoms of fatigue are the expression of a change in the ratio between the available energy (the energy in an active living state at the moment) and the resistance which that energy has to overcome. If the task in hand is intrinsically uninteresting, the energies brought into play, liberated within the organism, or transformed from the potential to the active state, are small in quantity; therefore a small amount of work, producing a small increase of the resistances to be overcome, suffices to raise the ratio of resistances to energies above the normal, and, therefore, to produce the symptoms of fatigue; whereas, if the circumstances are such as to bring into play greater stores of the potential energy of the organism, a much greater amount of work may be done by the organism (whether bodily or mental work), before the ratio of the resistance to the available energy rises to the point at which symptoms of fatigue appear.

Reflection upon these familiar experiences yields then two propositions which, I believe, may be two master-keys to all the problems of fatigue—at any rate, to those of fatigue of the nervous system. They may be briefly stated as follows:—

(1) Fatigue of the nervous system is a state in which the ratio of the resistances to the active energies is raised above its normal value, either through increase of the magnitude of the resistances or through diminution of the quantity of disposable energy.

(2) The function of the resistances is essentially the limitation of activity: they are thus protective of the energy of the organism; they prevent the organism exhausting its store of potential energy through unduly prolonged, intense, or widespread activity.

If these propositions are well founded we must, in order to understand fatigue, have good working conceptions on the one hand of the sources of the energies concerned and of the conditions and nature of their operations in the nervous system, and on the other hand of the nature and seat of the resistances that limit and preserve these energies.

It has recently been contended, notably by Bethe [1] in Germany and by Deschamps [2] in France, that the nervous system is not the seat of the liberation of any energy, or at any rate that such liberation is no part of its specific functions, but that it merely distributes the active energies supplied to it by *stimuli* of all kinds and by the various organs of the body. I think most physiologists will hold that this view is quite unacceptable, and that the activity of the central nervous system does essentially and always involve liberation of energy, transformation of energy from the potential to the active form, and a consumption or using up of stores of potential energy. Verworn [9], who has made himself the protagonist of this latter view, seems to me to have proved it by his own experiments and those of his pupils, although, even without these particular experimental results, there was hardly room for doubt about the matter. Further, Verworn's experiments on the spinal cords of frogs confirm the view (about which also there was little room for doubt apart from these experiments) that fatigue may be produced by either of two processes, and that it commonly is produced by their conjunction—namely, (1) the process of accumulation of waste products of metabolism, CO_2, lactic acid and other substances; (2) the using up of stores of potential energy through excess of katabolism over anabolism. The former is called by the Germans "Ermüdung," the latter "Erschöpfung," and some English writers have proposed to use the words fatigue and exhaustion in these two senses, as the equivalents of these German words. I do not think we can profitably specialise these words in this way;[1] but, if not, we must have new words, for there can be little doubt that both processes take part in the production of fatigue, and we must distinguish between them and hold fast the two conceptions.

The question then arises—What are the seats within the nervous system of these two processes, "Erschöpfung" and "Ermüdung"? Verworn's view may perhaps claim the adherence of a larger number of physiologists than any other. It is very simple. According to this view, the cell-bodies or "ganglion cells" are the seats, or at least the principal seats, of both processes; and of both processes the essential result, which determines all the symptoms of fatigue, is a diminished katabolism and output of energy by the cell-bodies affected. "Erschöpfung" is thus, according to this view, a strictly local process, affecting only the bodies of the nerve-cells involved in any activity; while "Ermüdung" is both local and general, because the cell-body is liable to be poisoned not only by the waste products of its own activity, but also by those of the metabolism of other nerve-cells and of other tissues of all parts of the body, especially the muscular tissues; for these waste products, wherever formed, may pass into the blood and lymph and so may reach the "ganglion cells."

Now I venture to say that this picture of the nature of fatigue of the nervous system is very much too simple; the conceptions are, I contend, true and useful, but inadequate to the explanation of the complex manifestations of fatigue which meet us as soon as we consider the organism as a whole; and they are inadequate also for the explanation of many of the special and local manifestations of fatigue.

The picture needs, it seems to me, to be complicated by two most important additions. First, Verworn's scheme takes little or no account of the resistances that the nervous energies have to overcome. Now, if with him we regard the cells as seats of explosive decompositions, and the nerve-fibres as freely conducting excitations (either purely physical processes or, with more probability, as physico-chemical processes involving metabolic changes similar to those that occur in the cell bodies), then it is obvious that there must be points or parts of the nervous tracts which delimit functional units and groups of units; else, instead of the orderly sequence of nervous excitations, which normally results from any stimulation, the excitation would spread indefinitely throughout the nervous system, soon producing incoördinate general activity and a rapidly ensuing general exhaustion; in the way the results of which we see in the convulsions of strychnine poisoning.

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[1] Because we need the word "fatigue" to denote the state of diminished efficiency of the organism as a whole commonly produced by these two processes in conjunction, and we need the word "exhaustion" to denote the extreme degree of the state resulting from excess of katabolism over anabolism.

Now the question, Where, in what structures, do these *resistances* reside? is one of secondary importance perhaps, so long as we recognize their nature and function. Still it is a question of some interest. In a recent paper Verworn recognizes the existence of resistances, and assigns them, like all the other peculiarities of the central nervous system, to the cell-bodies [10]. To that view serious objections may be raised. To mention only one, it is difficult to suppose that the cell-bodies are the seats both of explosive decompositions that liberate energy and also of the resistances that delimit the paths by which the excitation spreads towards the efferent nerves. On the other hand, there are many considerations tending to show that the principal seats of these resistances may be the synapses or places of junction of the neurones.[1]

If we assume that the synapses are the seats of the resistances, and that the process of transmission of the impulse across the synapse is one that results in a temporary raising of the resistance of the synapse, then we have a conception which enables us to account satisfactorily for many manifestations of fatigue. I will point out a few of the facts which seem to point to the synapse rather than to the cell-bodies as the principal seats of the increased resistance. It has long been notorious that nerve-fibres are but little, if at all, liable to fatigue; and there is good reason to believe that the cell-bodies also are fatigued, not easily or rapidly, but only under prolonged activity or when deprived of the normal means of recuperation—deprived, that is, of the normal blood-supply. Yet we have evidence that local fatigue may be rapidly induced, and may as rapidly pass away, in the various levels of the nervous system.

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[1] See especially Sherrington's "Integrative Action of the Nervous System," Lecture I. In spite of all that has been written of the synapse in recent years, one still finds widely accepted the following argument: The nervous system consists of cell-bodies and nerve-fibres, therefore all those of its properties that are not displayed by peripheral nerves are due to the cell-bodies. Verworn himself still uses this argument, and so little is the conception of the synapse understood that Verworn, in a recent paper, interprets Sherrington as meaning by the term "synapse" the place of junction of the axis cylinder and the cell-body of a single neurone. That, I take it, is implied when he writes, "Goldscheider und Sherrington denken an die Uebergangstelle des Nerven in den Ganglionzellkörper, an die 'Synapse,' wie Sherrington sich ausdrückt" (*Op. cit.*, p. 134). Sherrington inclines to the view that the process of transmission of the excitation across the synapse is a purely physical one (*Op. cit.*, p. 17); but if, as he also inclines to believe, it is a process very readily affected by the presence of toxic substances in the blood, and if it has the effect of immediately and temporarily raising the synaptic resistance (fatigue), and of ultimately leaving it (when the fatigue effect has passed away) permanently diminished (habit), these effects would seem to demand for their explanation the assumption of some metabolic activity at the synapse. In a paper in *Brain* (vol. xxiv.) I have adduced other arguments in support of the assumption of intercellular substances, the seats of synaptic metabolism of a highly specialized kind.

Such local fatigue occurs in the reflex arcs of the spinal cord, if a stimulus is continuously applied to the afferent path [8]. It occurs in the motor region of the cerebral cortex if one spot be continuously or repeatedly stimulated [4]. It occurs still more readily in the higher levels of the brain concerned in perception—*e.g.*, in all the cases of ambiguous figures which may be perceptually interpreted in two or more different ways, and of which the different modes thrust themselves alternately upon consciousness. I have studied these alternations and shown reason to suppose that a rapidly induced local fatigue of the higher cortical paths concerned is the principal condition of this alternating activity [5]. In the same way the extreme liability to fatigue of the paths of the higher cortical levels seems to underlie the extreme instability of all our thought processes; the fact that our attention can never be held fixedly and unchangingly upon any single feature of an object, but always, in spite of all voluntary efforts to the contrary, passes on from feature to feature, or plays to and fro over the object of thought.

In all these cases we seem to be dealing with a rise of the resistance of a nervous path rapidly induced by its activity and as rapidly passing away; a rise of resistance which serves to divert the excitation-process from the path to some other channel, and so to prevent the injurious effects of a too-prolonged activity of any one path.

These are manifestations of local fatigue, of increase of the ratio of resistance to energy, induced not by diminution of energy, but by increase of strictly local resistance.

Again, experiments with the ergograph have been used much in the study of fatigue, but one interesting feature of such experiments has been unduly neglected, I think. I draw attention to it in this connection, because it seems to bear out the view I am setting forth.

The subject of the experiment has to lift a heavy weight repeatedly at brief intervals, by bending as far as he can his middle finger. Fatigue very soon manifests itself in the form of a diminished extent of the bendings of the finger. Now, if you carefully observe the subject as he continues to repeat his efforts, you will usually see that, while at first he contracts only the muscles immediately concerned in the flexion of the finger, other muscles come into play as the flexions become diminished in extent; first the muscles of the upper arm, then those of the shoulder, then those of the trunk, still later the muscles of the lower limbs and of the arm and shoulder of the opposite side, and even those of the face, jaw, and neck on both sides. There takes place, in short, a spread or march of the excitation (not unlike the march of Jacksonian epilepsy) from the motor tract directly concerned in producing the flexion of the finger to adjacent motor tracts, then to successively more distant tracts.

Now this is just what must occur if, while the supply of liberated nervous energy is maintained at a high level of potential, and while the subject continues to direct it towards the one set of muscles, the resistances of the paths through which the excitation reaches those muscles are liable to be rapidly raised owing to the intensity of the process and to its repetition at brief intervals. The excitation will overflow, first into the most nearly connected tracts; later into those successively more remote, as the resistances of the more nearly connected tracts become in turn increased.

A very brief rest suffices to abolish or to diminish in very large degree any fatigue symptoms induced by ergographic work; and if in the ergographic experiments the intervals between successive bendings of the finger are made equal to several seconds, the evidences of fatigue are much diminished.

We have then abundant evidence of the rapid production of local increase of resistance in the central nervous system, and all that we know of the cell-bodies and of the axis cylinders makes it improbable that they can be the seats of these rapidly induced and very transitory local increases of resistance. Further, the fact that these local increases of resistance seem to be most rapidly induced in the higher levels of the central nervous system, where the connections between neurones must be regarded as least intimate and fixed, and to be least rapidly induced in the reflex arcs of the spinal cord, where the connections between afferent and efferent neurones are most firmly established, is in harmony with the view I am defending, and seems unintelligible if the alternative view is adopted.

It seems probable that the resistance of the synapses is liable to be temporarily increased not only locally by the transmission of the nervous excitation across them, but also generally by the influence of the waste products of metabolism brought to them in the blood, for they seem to be easily affected by chemical influences of many kinds.[1]

If so, then such action of waste products upon them must play a large part in that general increase of the ratio of resistance to energy which constitutes general fatigue, and which manifests itself subjectively in general tiredness and in sleepiness; and also in the onset of sleep or general quiescence of the brain, the most important of the modes in which the organism protects itself against exhaustion.

If we provisionally accept this view, that the synapses are the principal seats of resistance, and that their resistances are liable to be thus increased both locally and generally, then the simple picture of fatigue which I have associated with the name of Verworn becomes considerably complicated. In addition to local "Erschöpfung" of the neurones, and to local and general "Ermüdung" of them by waste products, the former of which means local, and the latter both local and general, diminution of energy supply, we recognise both local and general increase of synaptic resistances. We have then, I think, a scheme much less inadequate to the explanation of those familiar and important peculiarities of fatigue to which attention was drawn in the opening paragraphs.

But there were, I said, two complications to be added to the simple Verworn picture. We have added the resistances varying from local and from general causes. The second complication concerns the sources of the energies involved in bodily and mental activity.

According to the Verworn scheme every cell-body is, I take it, to be conceived as capable of generating or transforming or liberating a certain quantity of energy, which, up to a certain upper limit, varies with the intensity of the stimulus applied to the cell; and this energy is to be regarded as finding its field of operation wholly within the neurone or the particular small functional group of neurones within which it is liberated; and all neurones are of appreciably equal value in this respect.

But there is another conception of the mode of operation of nervous energy which goes back at least as far as Descartes; the conception—namely, that the energy liberated by chemical change, by katabolic process, in one part of the nervous system may be conducted through the nervous channels and may operate in other parts of the nervous system. This may be called *the hypothesis of the vicarious usage of*

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[1] There is something to be said for the view that they are the seats of the primary and principal influence of various drugs, possibly of alcohol, chloroform, strychnine, and others.

nervous energy. Now it seems impossible to get the physiologists of the laboratory, the physiologists who are chiefly concerned with the organs rather than with the organism, to consider this conception seriously and on its merits. If they occasionally refer to it, it is only to put it aside contemptuously as a naïve survival from the dark ages. Yet those who are in the habit of dealing with the problems of the organism as a whole, the physician and the psychologist, constantly make use of this conception, for they find it impossible to make progress in the understanding of their problems without it.[1] That fact gives the conception a claim to a more serious consideration than it has commonly received from the physiologists.

The conception towards which the study of the organism as a whole points is that the neurones of the afferent side of the nervous system (including the cerebellum) constitute a great common reservoir of free energy, in which the head of pressure varies from moment to moment with the ratio of the in-flow to the out-flow, and on which all efferent paths may draw in turn when they come into activity.

As to the sources from which this reservoir of free energy is supplied, no doubt every one of its constituent neurones is capable of making its contribution, and especially all those directly connected with, and stimulated through, the sense-organs.

But we shall never understand the problems of fatigue, or any other of the great problems of the brain, until we adequately recognise certain special sources of supply. And the study of fatigue serves to bring into prominence the importance of these special sources of energy.

In psychology we speak of instincts or innate dispositions to action, and we recognise that the constitution of every human being comprises a certain number of such instinctive dispositions, and that, when any one of these is awakened, whether by sense-impression or idea, the subject experiences a state of emotional excitement, and an impulse, a desire or aversion, a strong conation or felt tendency towards some kind of action. We recognise that under the driving power of such an impulse the subject can achieve tasks, can put forth quantities of energy, such as are impossible for him in the absence of any such conative-affective excitement.[2]

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[1] The necessity of conceiving nervous energy in this way was urged as long ago as 1886 by Hale White (*Lancet*, vol. ii., 1886, p. 161; see also *Brit. Med. Journ.*, October 17, 1908), who proposed to use the word "neurorrheuma" to denote nervous energy so conceived. Among many others who have approved of this conception are Victor Horsley (Hughlings Jackson Lecture, *Brain*, vol. xxix., p. 448), and S. G. Sharkey (Presidential Address to the Neurological Society, *Brain*, vol. xxvii., p. 12).

[2] For a fuller discussion of the *rôle* of these innate dispositions in the maintenance of human activities, I may refer to my *Introduction to Social Pyschology.*

It is for the physiologist to discover, if possible, the neural seats and bases of these innate dispositions. Recent work by Pagano seems to indicate that they may perhaps be found in the basal ganglia of the brain [6].

But however that may be, it seems safe to assume that these powerful impulses, which we experience in conjunction with our emotional excitements, and which enable us to energise in a way quite impossible in their absence, have nervous correlates, and that the physiological correlate of such an impulse is a great liberation of energy, in some centre or part of the central nervous system, which energy augmenting greatly the disposable free energy of the brain, can be utilised in the performance of any kind of action undertaken for securing the satisfaction of the impulse: and it seems that such an accession of energy can overcome the increased resistances obtaining in a state of moderate general fatigue, abolishing more or less completely in so doing both the objective and the subjective symptoms of fatigue; because the great accession of energy lowers the ratio of resistance to energy.

It is the possibility of these sudden accessions of energy that has rendered wellnigh futile all the many attempts hitherto made to obtain reliable objective measures of degrees of fatigue of the organism as a whole.[1] Rivers [7] has recently shown how great a disturbing factor in ergographic work is "interest," and the awakening of interest on the part of the subject means essentially the bringing into play of one or more of these special sources of energy.

Again, in such fatigue experiments as those of the Kraepelin school, the fatigue effects are constantly complicated, obscured, and rendered doubtful by variations of interest or of impulse which show themselves in spurts of all sorts.[2]

It is only when we take into account these special sources of energy that we can begin to understand the way in which, under circumstances that powerfully evoke our native impulses, we can execute without fatigue tasks which in less favourable circumstances would render us prostrate with every symptom of fatigue.

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[1] Binet and Henri have shown the inadequacy of the various methods employed previous to the date of publication of their work—*La Fatigue Intellectuelle* (1898); and in a recent critical study of the principal methods Ellis and Shipe (*American Journ. of Psychology*) have arrived at the conclusion that none of those investigated by them are reliable.

[2] The Kraepelin methods, I may remark in passing, seek to avoid these disturbances by keeping interest at a minimum. But the human subject is not easily kept in such a state; he will become interested, if only in the approaching end of his task, and hence great irregularities. In view of these difficulties I have suggested a method of estimating fatigue, which follows the opposite principle and seeks to keep interest at a maximum throughout, the task set being of the nature of a sprint; and I venture to think that this is the sounder and more hopeful principle to follow: see "A New Method for the Study of Concurrent Mental Operations and of Mental Fatigue" (*British Journal of Psychology*, vol. i.).

James, in a recent lecture entitled the "Energies of Men" [3], has dealt, in his brilliant way, with this aspect of our problem; he brought forward many examples of the way in which, under conditions suited to bring their powers most fully into play and to sustain their interest at a maximum, men may achieve incredibly severe and sustained efforts. He quoted especially the case of Colonel Baird Smith, who conducted the siege operations before Delhi during the Sepoy Mutiny, showing how for months he hardly ate or slept, or rested in any way, but worked almost continuously at tremendous pressure without showing or feeling fatigue, and how then, when the end came, and the circumstances ceased to demand and excite his efforts, he collapsed, an inert, emaciated, almost lifeless invalid.

Such cases illustrate again my main points—namely, (1) that fatigue is protective; (2) that fatigue is relative, is the expression of a rise of the ratio between resistances and energies; and they show how, if the conditions are such as effectively to call into play all the great special sources of energy, this ratio may be kept from rising above the normal, until the whole organism approaches absolute exhaustion; whereas, on the other hand, under conditions of boredom, the ratio is very readily raised above the normal.

If, then, there is any truth in what I have said of these sources of energy and of the vicarious usage of energy, it follows that we have to recognize as a second condition of general fatigue, in addition to the presence of products of metabolism in the blood, a diminution of the disposable energy, due to a diminished metabolism of the neurones in general and of these special sources of energy in particular; and the possibility of general fatigue of this nature is, I think, especially important from the point of view of the physician.

The long continuance of the chronic fatigues with which the physician so often has to deal seems to show that, in many cases at least, these are not toxic fatigues (the only kind of general fatigue admissible under the too simple Verworn scheme). It seems to imply the exhaustion of some of the sources of energy, which, rendering the maintenance of a due general pressure or potential of free energy impossible, causes the ratio of resistance to energy in all parts of the nervous system to remain at a high level.

In conclusion I would suggest that the conception of fatigue as an increased ratio of resistance to energy may possibly help us in arriving at an understanding of some nervous disorders.

A due balance between resistances and energies is essential to health, and it seems possible *a priori* that the balance may be disturbed by disorder primarily affecting either factor.

Suppose that, as an innate constitutional peculiarity, the resistances are deficient relatively to the energies in an otherwise normal or well-endowed brain; we should expect to see the possessor of such a brain an excitable sensitive person who, the great protective system being deficient, easily works himself to the point of exhaustion; exhaustion rather than fatigue will be his peculiarity. He is readily provoked to a great and rapid output of energy, and his brain does not easily return to rest; general excitement is slow to die away, and he finds difficulty in sleeping after any effort, for the condition of increased and predominant resistance throughout the brain is not easily attained; the system remains uselessly at work and wears itself out: there follows exhaustion secondarily developed, owing to the deficiency of the protective system of resistances. We should then get the typical picture of the hereditary or born neurasthenic; the disorder is brought on by any strenuous course of life, and its early stages are characterized by hyperæsthesias of all sorts, undue sensitiveness to all sense-impressions, general irritability or irritable weakness, insomnia, increased reflexes; the patient may have the power of working extremely effectively for a short time, but such work soon leads to exhaustion and leaves the patient incapable for a long time of again getting up a sufficient head of nervous pressure to renew his labours. It is, I think, in harmony with this view that some of our most brilliant and original intellects have shown marked neurasthenic tendency, *e.g.*, Charles Darwin and Herbert Spencer.

The neurasthenia induced by shocks—*e.g.*, the mental and physical shocks of a railway accident—may possibly be brought into line by the supposition that the brief and general over-stimulation of the nervous system, which was too sudden to allow the increase of resistances to play its normal protective part, has more or less paralysed the synapses, so that they no longer are capable of building up and maintaining the due resistances; and again irritable weakness dominates the scene.

In this way it seems to me we may perhaps account for the paradoxical character of neurasthenia, for its combination of excess and defect of energy, the difficulty of getting up steam, the easy and rapid running down of the effort, if an effort is successfully initiated.

There is another great type of functional disease—*hysteria*, so like and yet so different from *neurasthenia*, which seems to be also a form of chronic fatigue.

Here possibly the primary evil is a constitutional defect of the energies relatively to the normal resistances. Such relative defect is apt to be brought to light by any course of life that makes considerable demands on the nervous energies, and shows itself in the failure to maintain the unitary functioning of all the many neural systems and sub-systems that make up the brain. The systems tend to become functionally separated from one another, because the supply of energy is insufficient to overcome the resistances of the highest levels of the brain and to keep open the connecting channels; hence each neural system tends to function independently of the rest, and each, being removed from the reciprocal control and inhibition normally exerted by all the rest, may function in excess, or, on the other hand, may lie dormant. Hence we get the curious pictures of overactions combined with partial defects of action, of hyperæsthesia with anæsthesia, of local contracture and paralysis, of incapacity for effort and violent convulsions, of fixed ideas and of easily changed convictions, of extreme suggestibility and extreme obstinacy. We get the characteristic symptoms of mental dissociation,[1] and in extreme cases we get the falling asunder of the neural basis of unitary personality into two or more functional systems, each of which becomes more or less fixed in its isolation; and we thus get the most strange of all the functional disorders—the cases of divided or multiple personality.

REFERENCES.

[1] BETHE. "Zur Theorie der Zentrenfunktionen," *Ergeb. der Physiol.*, Bd. v.
[2] DESCHAMPS. "Les Maladies de l'Energie," Paris, 1908.
[3] JAMES. "The Energies of Man," *Philosophical Rev.*, 1907.
[4] LEVY. "An Attempt to Estimate Fatigue of the Cerebral Cortex," *Journ. of Physiol.*, vol. xxvi.
[5] MCDOUGALL. "Physiological Factors of the Attention Process," *Mind*, N.S., vol. xv., p. 340.
[6] PAGANO. *Archiv. Ital. de Biol.*, 1906.
[7] RIVERS. "The Influence of Alcohol and other Drugs on Fatigue," London, 1908.
[8] SHERRINGTON. "The Integrative Action of the Nervous System," London, 1906.
[9] VERWORN. "Die Vorgänge in den Elementen des Nervensystems." *Zeitschr. f. Allg. Physiol.*, Bd. vi.
[10] *Idem.* "Die cellular physiologische Grundlage des Gedachtnisses," *Zeitschr. f. Allg. Physiol.*, Bd. vi.

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[1] In this connexion I would refer the reader to my article "The state of the Brain during Hypnosis" in vol. xxxi. of this Journal.

THE PATHOLOGICAL EXAMINATION OF TWO CASES OF AMYOTONIA CONGENITA WITH THE CLINICAL DESCRIPTION OF A FRESH CASE.

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THE object of this paper is to place the pathological nature of the malady first described by Oppenheim in 1900, upon a firm basis, and to emphasize certain clinical features of the disease which are peculiar and which, in our opinion, demand the recognition of the condition as a separate clinical entity, not to be included among the hitherto described maladies which are rightly grouped together under the term myopathy. Since Oppenheim first described the disease in 1900, though many cases have been recorded from the clinical side, there have been but three pathological examinations, and of these three only one can be said to have been complete. Spiller in 1905 was the first to make a pathological investigation upon a well-marked case at the age of 22 months. He found no changes in the nervous system, but a condition of intense regression in the affected muscles, and, in addition, a pathological state of the thymus gland. Bing in 1907 removed a portion of muscle from a patient aged 3½, and the changes that he found were comparatively trifling. Baudouin in 1907 made a complete and detailed examination of a patient aged 4 months. He found intense regressive changes and sclerosis in the affected muscles, and he was the first to point out the greatly hypertrophied muscle fibres which were seemingly not present in Spiller's case. Further, he found conspicuous changes in the nervous system, which comprised certain changes in the external group of the ventral horn cells, smallness of the ventral roots suggesting arrest of development and a corresponding abnormality in some fibres of the peripheral nerve-trunks. Sclerosis of the thyroid and of the thymus gland were present. Baudouin came to the conclusion that the sum of the anatomical changes was just such as is met with in cases of myopathy.

The results of our investigations upon the two cases here reported closely resemble the findings of Baudouin, and since the difference between the findings in Spiller's case and in Baudouin's case was a difference of degree rather than of kind, we may say that the four pathological investigations now on record are mutually confirmatory, with the one exception that the striking changes found in the thymus by Spiller, and in the thyroid and thymus by Baudouin, were certainly not present in one of our cases, and there was no clinical evidence of their presence in the other case. We suggest, therefore, that the changes in these glands were fortuitous, and that such changes play no part in the essential pathology of the disease.

In the interpretation of the pathological findings the exceedingly difficult problem arises as to the relative parts played by (1) arrest of development, (2) regression, and (3) regeneration, in the production of the histological changes. The fact that the investigations have been carried out upon subjects at widely different ages—namely, at 4 months, 22 months, 3 years and 7 years respectively, all these cases being congenital and not differing widely in the depth of the affection—suggests that the nature of the changes found at the different ages when the disease had been present for increasing periods would throw some light upon this question. As a matter of fact, however, the differences between the changes found at the various ages prevents us from coming to any conclusion upon this matter. For Baudouin's patient, who was only 4 months old, presented the most conspicuous changes of all the four cases; while in Spiller's clinically severe case, aged 22 months, the changes were the least marked; while the changes in our two cases, aged 3 years and 7 years respectively, were not more intense in the younger child, though clinically the younger child was far more severely affected than the elder. The objective pathological evidence seems in favour of increasing regressive changes without any regeneration; the clinical evidence suggests a developmental error in that the malady is almost always present at the time of birth, and it demands a pathology that admits of improvement, for not only is the history of progressive improvement common in the history of the recorded cases, but also the subsequent history of two of the cases that were published by one of us (James Collier) with Dr. Wilson in *Brain*, vol. xxxi, 1908, puts the question of recovery beyond doubt. In the first of these cases, a severe congenital case which had been under the close observation of Dr. Whait from the time of birth, slowly progressive improvement occurred up to the seventh year when the knee-jerks were obtained for the first time. After this more rapid improvement occurred, and the boy can now walk fairly well at the age of 9 years. In the second case complete recovery of the upper extremities, which were severely affected, has occurred with return of the deep reflexes in the upper extremities, while the lower extremities have improved greatly. At the same time we must point out that two cases which have come under our notice have shown progressive deterioration, and one of these is the case here reported. Our interpretation of our pathological findings allows of the possibility of improvement, but it argues forcibly for the occurrence of progressive deterioration in some of the cases.

The similarity of the morbid anatomy of amyotonia to that of the myopathies is certain, and does not admit of any discussion; but the clinical aspect of this disease is so widely different from that of any hitherto described form of myopathy as to warrant, in our opinion, the retention of a separate name for this distinct clinical entity. Although more than forty cases of amyotonia have been recorded, yet until quite recently no case had been brought forward in which there was any hereditary or familial tendency, or in which a familial association with myopathy had occurred. Sylvestri, however, has recently recorded two cases of undoubted amyotonia in which all these associations were manifest. These patients were the first and fifth children of a goitrous mother, the second and third children were healthy, and the fourth was a cretin. A maternal aunt suffered with the Landouzy-Dejerine form of myopathy. The elder of the two children presented well-marked amyotonia from the time of birth, and she progressively improved till the age of 16 years. During these years her condition had changed from one of "absolute flaccid paraplegia with paresis of the upper limbs" to one of "a certain degree of general asthenia." It is somewhat difficult to follow Sylvestri in his further description of this case, for he says that at the age of 16 years, "so far as could be judged by sight, she presented the most typical picture of Erb's disease, complicated with a form of progressive muscular dystrophy (juvenile form of Erb). The left deltoid, supraspinatus and triceps were hypertrophied, and the right brachialis anticus was pseudo-hypertrophied."

In the younger child the amyotonia was typical, all the voluntary muscles being severely affected with the exception of those supplied by the bulb. In neither of the cases was there any pathological examination. The familial incidence of these cases and their hereditary and familial association with myopathy do not controvert the fact that in all other recorded cases these associations have been absent. We do not think, therefore, that it is justifiable in the present state of our knowledge of this malady to depart from the opinion that one of the characteristic features of amyotonia is the absence of hereditary and familial tendencies and of associations with myopathy. This has been the opinion of nearly all writers upon this subject, and with it our own clinical experience of the disease is in accord.

We wish to emphasize in this place those clinical features of amyotonia which are widely different from those occurring in any form of myopathy. These are as follows:—

(1) The absence of any familial tendency in the incidence of the disease.

(2) The absence of any familial or etiological relation with myopathy.

(3) Amyotonia is in the majority of cases congenital. In a small minority of cases it appears suddenly and in fully developed form after certain acute diseases.

(4) The local wasting and weakness of an individual muscle, or of a group of muscles, that are characteristic of all forms of myopathy are not met with in amyotonia.

(5) Affection of the periphery of the limbs, and especially of the intrinsic hand muscles, which is the invariable rule in amyotonia, is of the greatest rarity in any form of myopathy.

(6) Amyotonia never spreads to regions previously unaffected. Slow spreading of the affection from muscle to muscle is a characteristic of all forms of myopathy.

(7) The deep reflexes are absent from the first in amyotonia. In myopathy they are present at first, and they slowly diminish and are lost as the affection of the muscles concerned increases.

(8) The deep reflexes may appear in amyotonia when improvement occurs and, having appeared, remain permanently. In myopathy the deep reflexes never reappear after disappearance.

(9) A majority of the recorded cases of amyotonia have shown a tendency to improve progressively, and this improvement may reach a stage of practical recovery.

Our best thanks are here expressed to Dr. Ormerod, under whose care one of the patients here reported was admitted into St. Bartholomew's Hospital suffering with a pneumonia which proved fatal; and to Dr. Langdon Brown for his kindness in providing us with the pathological material.

Clinical Details.

Case 1. (This case has already been recorded upon the clinical side by one of us [J. C.] with Dr. Wilson in *Brain*, vol. xxxi., 1908.)—S. A. I., a boy aged 5 years 3 months, was admitted into the National Hospital under Dr. Collier in February, 1907, with inability to walk and clumsiness of the upper limbs. The parents were healthy, and there was no history of family disease. There were seven children of the family, of which the patient was the fifth. Two of these had died in early infancy of blood-poisoning and of whooping-cough and convulsions respectively. The other children were healthy. The mother has had good health during the pregnancy, and birth was natural and easy. The child was breast-fed till 15 months old. From the time of birth the mother noticed that he could put his limbs into extraordinary positions, quite unlike her other children, and that his legs were very weak. He was late in attempting to stand, and never accomplished more than standing up when holding on to anything. He could never walk alone. His ability to stand when supported lasted only two months, when with increasing bodily weight he seemed to become weaker and weaker on the legs. On admission he was a pale, thin, starved-looking child of average height. His intelligence was unusually good for his age. The special senses and cranial nerves were normal. The facial musculature was well developed and symmetrical, and the facial movements were good. There was no difficulty in speaking and swallowing, and the tongue, which was protruded straight, was normal in size.

All the muscles of the upper extremity were below the normal size, but all voluntary movements could be performed with fair range and power. There was most absolute hypotonia of all the muscles, with marked relaxation of all the ligaments, so that the joints were all flail-like. The upper limbs could be folded behind the back and could be put into many extraordinary positions without causing the least pain. The muscles of the neck and of the trunk were thin and weak. He could only just raise the head from the bed when lying in the supine position, and he sat up with difficulty, the body being bunched up with a tendency to kyphosis. The respiratory muscles were unaffected. The muscles of the lower limbs were flabby, but they had a firmer feel than those of the upper limbs. There was slight contracture of the flexors of the hips and of the hamstrings, and a more decided contracture of the muscles of the feet, giving rise to a considerable degree of pes cavus. All voluntary movements were possible and were of fairly good range, except in so far as they were hindered by the contractures; and the power was fair, but the extensors were relatively weaker than the flexors. The excitability of the muscles to faradism was much diminished, and the child showed no signs of pain when strong faradism was applied. The reaction to galvanism was normal, sensibility was normal, and there was no affection of the sphincters. The superficial reflexes were normal; the deep reflexes were entirely absent.

Under treatment this patient improved considerably and gained the power to stand and to make an attempt to walk with assistance.

Two years after his admission into the National Hospital, and when aged 7, he was attacked with pneumonia and was admitted into St. Bartholomew's Hospital, where he died after a few days' illness. The autopsy was performed by Dr. Langdon Brown in the presence of one of us.

Case 2.—H. K., a little boy, aged 3½ years, was admitted into the National Hospital under Dr. Collier in June, 1908, for inability to stand and walk, and for weakness and limpness of the limbs which had been noticed since soon after birth. He was the fifth and youngest child of healthy parents, and the other children were strong and well. There was no history of any similar disease in any relations. The birth was natural and occurred at full term. From the time of birth his mother noticed that she could put his napkins on much more easily than those of her other children, because his legs went into the right position for this operation so easily, and because he did not kick like the other children. She noticed no other peculiarity till the child was a year old, when she tried to stand him up and found that the legs doubled up anyhow under him. He was never able to stand or to walk, but when 18 months old he learnt to creep round the table by wriggling his body about. As he got older and heavier he lost this accomplishment. He could sit up, but his back bunched up in so doing, and the least touch sent him over, and when over in any direction he could not of himself regain the sitting position. He could not turn in bed or move his legs from the position in which they were placed.

On examination the child was found to be well proportioned and, with the exception of some of the limb muscles, well developed and of good weight. He was very intelligent, and was forward for his age. The special senses, optic discs, and cranial nerves were not affected. There was no affection of the muscles of the face and jaw, and swallowing was normal. There was no obvious wasting of the muscles of the neck, but these were obviously very weak, for the child never sat with his head upright, but it fell about in any direction. The muscles of the back and of the abdomen were very weak. There was a marked general kyphosis of the spinal column in the sitting position, which at once straightened out if the child were raised by the shoulders. He could not raise himself when lying down nor turn in bed. The diaphragm and intercostals acted well, as seems to be the invariable rule in this disease. In the upper extremities the muscles had a peculiar soft, homogeneous feel and could not be differentiated by touch from the overlying skin and subcutaneous tissues. They were small, but they were equally small throughout the limb, and there was no local muscular wasting, such as is always present in the true myopathies. Passive movements at all the joints were very free. Considerable hyper-extension was possible at the elbow-joints, and this was very conspicuous at the wrist- and finger-joints. If the arm was held at the elbow and shaken the flail-like movements of the hand and fingers were striking. The power in the upper extremities was very poor, and he could make few useful movements with the hands. In the lower extremities the muscles were even smaller and softer than in the upper extremities, but there was no local wasting. There was apparently no volitional movement either at the hip or at the knee, but slight movements at these joints could be obtained reflexly by tickling the soles of the feet. All movements of the ankles and of the toes could be performed, but with very little power. The hypotonicity of the muscles of the thighs and of the calves was very pronounced, and the feet could easily be touched together behind the head. There was a little contracture of the calf muscles and of the hamstrings. The hyper-extensibility of the ankle (so that the dorsum of the foot is in contact with the front of the tibia in the position of rest) that has been a feature of so many of the recorded cases was not present in this case, neither were the feet conspicuously long and padlike.

The sphincters were not affected. The deep reflexes were persistently absent. The superficial reflexes were of normal type. Sensibility was unaffected. There was no lowering of sensibility to the faradic current, as has been observed in many of the cases. The child objected so strongly to the application of the faradic current that no satisfactory examination of the electrical excitability of the muscles was made, but it was obvious that the faradic excitability was much lowered in the affected muscles. The child remained in hospital for many months, and he was treated with careful feeding, massage and passive movements, in spite of which he was obviously weaker in the affected regions at the time of his discharge than he was when admitted. On the other hand, there was no extension of the muscular weakness to regions previously unaffected. With the consent of the parents a portion of the hamstring muscles was removed from the right thigh under general anæsthesia, and an account of the changes that were found in these muscles, follows in the pathological description. It must be here pointed out that no abnormality of the thyroid gland or of the thymus was clinically detectable, and that there was no œdema present at any time.

Remarks.—This was a typical case of amyotonia congenita of somewhat severe degree. The distribution of the affection followed that of the classical cases. The degree of affection of the thigh muscles, in which paralysis was practically complete for volitional movements, was extreme and unusual, and such severity of muscular affection has not been previously recorded. The pathological examination of the muscles was made when the disease had existed for rather more than three years, as compared with seven years in Case 1. This is one of the only two cases that have come under our observation in which progressive deterioration has occurred. In all our former observations there has been progressive improvement.

Pathological Examination.—The morbid changes found in the portion of muscle excised from Case 2 may be dealt with first, as in this case an earlier stage of the disease is represented. When the biceps cruralis was exposed at the operation for the removal of the muscle it appeared remarkably pale and was evidently much infiltrated by fat; in fact, very little muscular tissue could be recognized by the unaided eye. The portion excised was immediately placed in Zenker's fluid for fixation and hardening, and then, after dehydration, it was embedded in celloidin and sections 10 μ thick were easily obtained. Small pieces of the muscle were, in addition, mordanted in Müller's fluid in order that the myelinated intramuscular nerves could be examined by Weigert's method. The sections of the celloidin material were stained chiefly with hæmatoxylin and Van Gieson's picro-fuchsin.

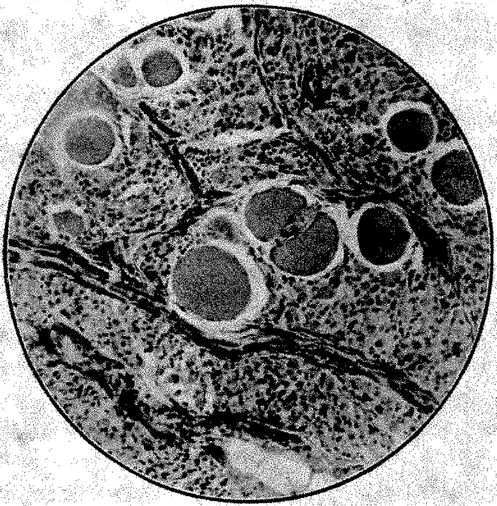

FIG. 1.

Photomicrograph of a transverse section of the muscle. It shows (1) irregular small atrophied fibres; (2) enlarged or hypertrophied fibres which are generally circular in outline; (3) dense strands of connective tissue that run through the muscle.

Muscle Changes.—A few bundles of normal fibres were found in the piece of muscle excised, but they occupied only a very small proportion of the cross-section. The most prominent change was an extreme smallness of the muscle fibres. In a majority of the muscle bundles no a single fibre approaching the normal in size could be seen; indeed, their average diameter was from 6 to 12 μ, though a few of the small fibres exceeded this considerably. The contour of these small fibres in cross-section was irregular and, as a rule, oval or round; even where they were densely packed together they never presented the polygonal or facetted appearance which characterizes the normal skeletal muscle fibre when seen in cross-section. In specimens in which these small fibres were cut longitudinally they were seen to be fairly uniform in calibre and their cross-striation was well preserved. In many of the small fibres there were scarcely more nuclei than may be found in normal adult fibres, but in some there was a marked proliferation of

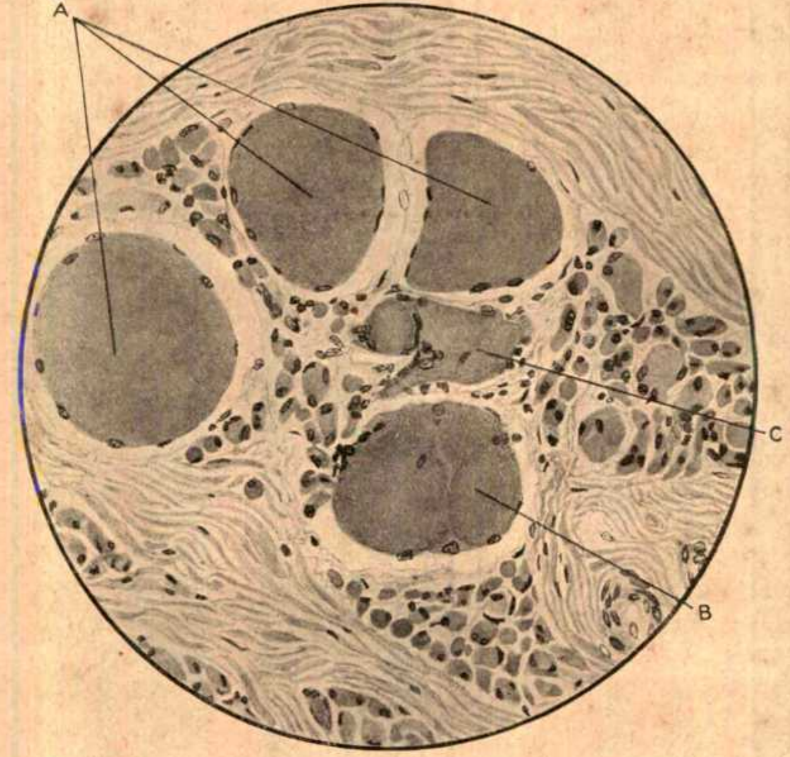

FIG. 4.

A drawing of a transverse section of the muscle: it shows the irregular size and shape of the smaller fibres, and that in many of them the sarcolemmal nuclei are proliferated. *A*, three hypertrophied fibres that are apparently normal in structure and contain only peripheral nuclei. *B*, an enlarged fibre, containing central nuclei, which is beginning to divide. *C*, an enlarged fibre that is already partly broken up.

the sarcolemmal nuclei, and in places this was so great as to conceal the substance of the fibre. No degenerative or regressive changes could be made out in these small or atrophic fibres.

A still more striking feature of the appearance of cross-sections of the muscle was the presence of numerous relatively enormous fibres which were generally of circular outline in cross-section, and which measured from 100 to 150 μ in diameter. These greatly enlarged fibres generally lay isolated in or among the bundles of small fibres; they were only rarely found forming bundles of themselves. Apart from their size and their unnaturally cylindrical shape, the greater number of these giant fibres appeared to have a normal structure and contained nuclei only immediately under the sarcolemmal sheath. Their cross-striation was distinct, but in many of them the longitudinal striation was unduly prominent. In a certain proportion, however, of these giant fibres unmistakable regressive changes were present; some of them contained central nuclei either embedded in normal myoplasm or surrounded by a zone of undifferentiated sarcoplasm. Occasionally the muscle tissue immediately surrounding the central nuclei had a remarkable tendency to take a deep but diffuse stain with hæmatoxylin. In other fibres the regressive process had gone still farther and lead to central vacuolation and splitting up of the fibre, while in some the myoplasm was more or less disintegrated and invaded by the

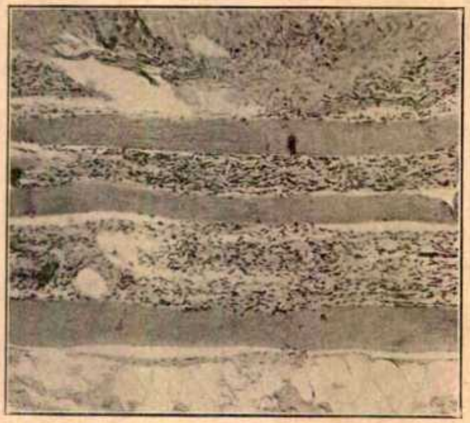

FIG. 2.

Photograph of a longitudinal section containing three hypertrophied fibres, separated by small or atrophied fibres in which there is a considerable increase of nuclei.

sarcoplasm and sarcoplastic nuclei. In longitudinal sections portions of these fibres could be seen transformed into protoplasmic masses pervaded by nuclei, the sarcolemmal sheath generally remaining intact. The central vacuolization resulted, in many fibres at least, from the fusion of a series of small discrete vacuoles in the walls of which nuclei were frequently present. In longitudinal sections it could be seen that a few of the enlarged fibres were splitting up longitudinally into apparently well-formed fibres of small calibre, provided with a normal sarcolemmal sheath and nuclei (fig. 5).

The muscle was also characterized by the great increase of connective tissue which was arranged both in massive bundles running between and surrounding the muscle bundles, and in very delicate strands of connective tissue which in places even penetrated between the individual muscle fibres. There was also a notable determination of loose fatty tissue between the muscle bundles, and to this the remarkably pale naked-eye appearance of the affected muscle was due. The muscle spindles were on the whole normal, but in places their sheaths appeared slightly thickened.

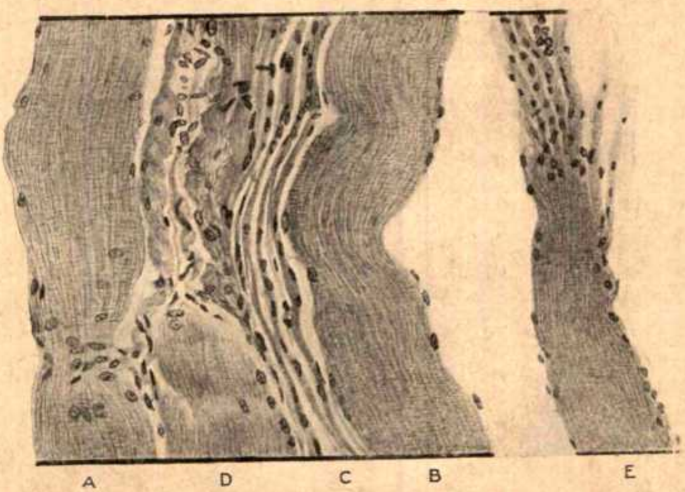

FIG. 5.

Drawing of a longitudinal section. *A* and *B*, hypertrophied fibres which are normal in structure; *D*, one that contains central nuclei and is breaking up by longitudinal fission. *C*, slender atrophied fibres. *E*, an hypertrophied fibre that is splitting longitudinally.

Considerable thickening of the walls of many of the blood-vessels was a noticeable feature of many of the sections. This was the result chiefly of a periarteritis, but there was also some proliferation of the intimal cells. A few foci of small round-celled infiltration were found, which generally lay in immediate relation to a vessel, but these were only rarely met with in a large number of sections examined.

In the sections of muscle stained by Weigert's method there seemed to be remarkably few myelinated nerves, and the myelin sheaths of those that were present seemed poorly developed.

In Case 1 death occurred from pneumonia, and a complete autopsy was made. There was no abnormality of the thyroid, thymus, or spleen found, and, with the exception of the lungs, the internal organs appeared natural; the muscles of the affected regions were small and very pale, and contrasted very sharply with the muscle of the diaphragm, which was not affected. The muscles were fixed in Zenker's fluid, the peripheral nerves in Müller's fluid, and the central nervous organs as well as the other organs were placed in a 10 per cent. solution of formalin in normal saline solution.

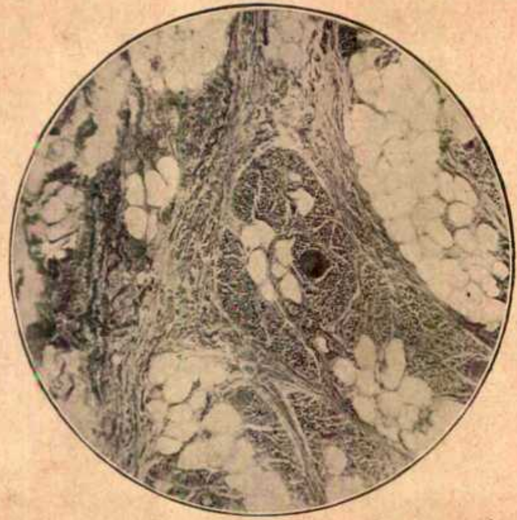

FIG. 3.

Photograph under a low magnification to show the excess of fat and connective tissue the muscle contains, as well as the small size of its fibres; one hypertrophied cylindrical fibre lies in the centre of the figure.

Muscle Changes.—The changes in the muscles were very similar to those found in the preceding case. The most striking difference was a greater proliferation of the sarcolemma nuclei in the small fibres, many of which were almost concealed by continuous columns of nuclei arranged along them. Further, in many places sarcoplastic nuclei could be seen lying apparently free in the connective tissue. The large hypertrophied fibres so conspicuous in the other case were much less numerous and less prominent in this case. There was also a greater amount of connective tissue in the more severely affected portions of the muscles, and it was more densely sclerosed. In places, too, there was a remarkable deposition of fat, so that portions of the muscle seemed to be completely replaced by fatty tissue.

The pathological changes, however, varied considerably from muscle to muscle, and even in different portions of the same muscle: the diaphragm, for instance, was unaffected, and the biceps brachialis was much less severely affected than were the pectoralis major and the semitendinosus. This indicates a definite parallel between the degree of the clinical symptoms and that of the pathological changes in the muscles.

Peripheral Nerves.—The external popliteal and the median nerves were preserved for microscopical examination. In sections stained by Weigert's method the nerve fibres in cross-section seemed rather far apart, and the myelin sheaths of a small proportion of the fibres were poorly developed. On macroscopical examination it was obvious that the ventral spinal roots throughout, but more especially those of the lumbo-sacral region, were remarkably slender in relation to the size of the dorsal roots, and this appearance was confirmed by their examination in Weigert preparations; for under the microscope their fibres appeared not only much reduced in number, but individually slender and poorly myelinated.

Spinal Cord.—The white matter of the spinal cord showed no abnormalities and the fibres were well myelinated. On the other hand, Nissl preparations revealed unmistakable pathological changes in the ventral horns at all levels of the spinal cord. The large cells of the motor type were much reduced in number, in places there appeared to be scarcely one-third of the normal number of these cells present, and those that were present were individually smaller and more angular and irregular in shape than are normal cells. These changes were found in all regions of the spinal cord, but they were most conspicuous in the lumbar and sacral segments and in the lower segments of the cervical enlargement. In the cervical enlargement it was the lateral and the postero-lateral groups of cells that were the most severely affected. The cells of the dorsal horns and those of Clarke's column were normal.

It is now necessary to compare these pathological changes with those that have been already discovered in similar cases. But till now only Bing, Spiller, and Baudouin have had the opportunity of recording such observations. These have been already abstracted and analysed by one of us with Dr. S. A. K. Wilson [4].

Bing's [3] examination is the least important, as he had only a very small piece of muscle removed during life, and in its fibres he found only an increase of nuclei.

Spiller [6], who made a *post-mortem* examination on a case that died at the age of 22 months, found the muscles pale and infiltrated by fat, the muscle fibres small, and a considerable increase of the nuclei of the connective tissue. The peripheral nerves, the dorsal and ventral spinal roots, and the central nervous system were normal. The report, especially of the muscle changes, is not as complete as could be wished.

Baudouin's report [2] is much fuller. In a child that died at the age of 4 months the muscle was largely replaced by fat and contained a considerable excess of connective tissue; the majority of the fibres were atrophied and contained a large number of nuclei, but others were enormously hypertrophied, measuring up to 100 μ in diameter, and were often penetrated by sarcoplastic nuclei. Some of these hypertrophied fibres were found splitting longitudinally. There was also a considerable diminution in the size, and perhaps in the number too, of the ventral horn cells, and the ventral roots of the spinal cord were less than half the normal size and were poorly myelinated.

From these short abstracts it is evident that the pathological changes in the muscles in Baudouin's case were practically identical with those present in the two cases we have been able to examine, and there was an equally striking similarity between the alterations in the peripheral nerves and the ventral horns of Baudouin's case and in our case that came to *post-mortem* examination. The changes Spiller found in the muscles do not differ essentially from those we found, though he does not refer to the presence of hypertrophied fibres. In the nervous system, on the other hand, he found no pathological changes. These discrepancies may be perhaps explained by a difference in the intensity or the stage of the disease.

There is not much difficulty in interpretating these muscle changes. The most common, constant, and characteristic change was the smallness of the majority of the muscle fibres. This smallness may be, on the one hand, attributed to a non-development of these fibres, or on the other to regressive or atrophic processes affecting them. The latter seems the more likely explanation, as the extraordinary irregularity in the size and shape of the fibres could be hardly expected if the smallness was merely due to an arrest of development; and the considerable increase in their nuclei and the presence of apparently free nuclei in the connective tissue are much more in favour of the view that they arise by the regression of normal fibres. Further, it must be remembered that in places we found small fibres arising by the longitudinal fission of large fibres. Finally, the appearance is not that of undeveloped fibres; it is only towards the end of the sixth month of intra-uterine life that the central portions of the skeletal muscle fibres become differentiated and that the nuclei move to the periphery of the fibre, and they receive their sarcolemmal sheaths only at the time of, or shortly before, birth. But in all the muscles we have examined the central portions of all the small fibres were fully differentiated, and they possessed well-developed sarcolemmal sheaths.

The significance of the large hypertrophied fibres is more difficult of interpretation; it has been much discussed in relation to the myopathies, in which there are found similar large fibres certainly identical with those we describe here. Erb regarded the increase in size as a true hyperplasia preceding atrophy, while Dejerine has attributed it to a vicarious functional hypertrophy. Whatever may be their nature, the active regressive changes we have already described in them must be regarded as unequivocal evidence of a progressive pathological process.

The clinical features of the disease are easily correlated with the pathological changes we have found. The great atrophy and the decrease in number of the muscle fibres sufficiently explain the palsy, while the infiltration of the muscles by fat and the increase of connective tissues in them concealed in part the general atrophy. The atony may be attributed to the same cause, while the contractures that occasionally develop are evidently due to sclerosis of the fibrous tissue that is laid down in the muscles.

The fact that some cases improve has been regarded as one of the features of the disease. From the pathological point of view, however, improvement appears unlikely; we detected no evidence of regenerative or reparatory changes in the muscles, but the possibility that the atrophic fibres may regain their normal size and function must be admitted. But for the muscle to regain its proper function it appears necessary that the excess of fat and the fibrous tissue that is found, both in thick bundles and in fine strands, between the affected fibres should be absorbed or removed. This certainly does not appear likely.

In this connection we must again refer to the active regressive changes that were present in a considerable proportion of the enlarged fibres.

Finally, there remains to be considered the relation of the morbid anatomy of these cases to that of the myopathies. On comparing the muscle changes found in these two conditions, we must admit that there is no essential difference, though in our younger case there was, perhaps, not so great an increase of nuclei in the atrophied fibres as is generally present in the myopathies. Myopathic muscles are characterized by the presence of atrophied and hypertrophied fibres, with a proliferation of their sarcoplastic nuclei and various regressive changes in them, an increase of connective tissue and a deposition of fat; and it is exactly the same changes we record above. Further, we observed a thickening of the vessel walls which is almost constant in myopathic muscles, and the collections of small round cells which have been referred to are similar to those that may be seen in myopathic muscles.

Baudouin came to the same conclusion, for he wrote of the muscular alterations in his case as "les lésions de la regression musculaire telles qu'on peut les voir dans les myopathies."

It must be pointed out that the spinal changes observed by Baudouin and in one of our cases do not constitute an essential difference from the pathology of the primary myopathies, for one of us [5] has recorded a case of the juvenile form of muscular dystrophy in which practically identical spinal changes were present, and in that paper there are collected together a considerable number of similar cases that had been previously reported. Ballet and Laignel-Lavastine [1] have more recently published a similar observation.

REFERENCES.

(A complete bibliography of the subject will be found under Reference No. 4.)

[1] Ballet et Laignel-Lavastine, "Myopathie Ancienne," *L'Encephale*, 1908, Ann iii., p. 229.

[2] Baudouin, "La myatonie congenitale," *La Sem. méd.*, 1907, t. xxvii., p. 241.

[3] Bing, "Ueber atonische Zustande der kindlichen Musculatur," *Med. Klin.*, 1907, Bd. iii., S. 10.

[4] Collier and Wilson, "Amyotonia Congenita," *Brain*, 1908, vol. xxxi., p. 1.

[5] Holmes, "On the Spinal Changes in a Case of Muscular Dystrophy," *Rev. of Neurol. and Psychiatr.*, 1908, vol. vi., p. 137.

[6] Spiller, "General or Localized Hypotonia of the Muscles in Childhood." *Univ. Penn. Med. Bull.*, 1905, vol. xvii., p. 342.

[7] Sylvestri, "Contributo allo studio della Myatonia Congenita." *Gaz. degli Ospedale*, 1909, vol. xxx., p. 577.

AN EPIDEMIC OF ACUTE POLIOMYELITIS.[1]

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EPIDEMICS of acute poliomyelitis have seldom been recorded in England; in fact, of thirty-two epidemics described in medical literature, two only occurred in this country. The records of Swedish epidemics show that poliomyelitis must be far commoner in some other countries than it is in England. But, apart from a greater frequency of the disease, the segregation of the population of this country in large towns makes it more difficult to define the epidemic nature of the disease. Thus, although a few years ago it was well recognized that a large epidemic was occurring in London, the great difficulty in collecting the different cases rendered abortive all attempts to trace the epidemic. Although it would be impossible to add to the wonderfully painstaking descriptions of the Swedish scientists, yet we believe that an account of even a small epidemic in England is worthy of notice, in that it brings the disease in England into line with the disease as it occurs in other countries, and affords the opportunity of comparing its similarities and differences.

Thirty-two epidemics of poliomyelitis have been recorded, the first (an epidemic of eighteen cases) by Bergenholf, in November, 1881 [11]. The first large epidemic was reported by Caverly in 1894 [4]. It consisted of 136 cases, and occurred in an area of 15 miles in Rutland. It occurred during a very hot summer and was associated with cases of paralysis among cattle and fowls. Eighteen patients died, and in six cases there were constitutional symptoms without any paralysis. Unfortunately no autopsies were performed, and it was considered by some authorities to be epidemic cerebrospinal fever. There was, however, no purpuric eruption, and it seems (judging by later epidemics) to have been undoubtedly acute poliomyelitis.

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[1] My best thanks are due to Dr Dunlop, of Upminster, under whose care were most of these patients, for the kind way in which he aided me in tracing this epidemic.

Of the two epidemics recorded in England, one, recorded by Buzzard [3], consists of four cases. Two sisters living in the same house were attacked within six days of one another, and also two other children (who were not examined) living in the same street.

The other epidemic was recorded by Pasteur [12], and is of the greatest interest. In July, 1896, the whole of a family of seven children were attacked with fever within ten days; in three cases the fever was followed by paralysis, and by other nervous symptoms in the other four cases. The eldest child, Herbert George, aged 11, five days after the onset of the fever was paralysed in the left arm, and had a transient paralysis of the right side of his face and palate. The second child, Samnel James, aged 9, after seven days of fever was found to be suffering from rigid hemiplegia on the right side. The two next children, George, aged 8, and Edith, aged 6, had fever resembling that of the others, but did not develop any nervous symptoms. Alice, aged 5, after five days of fever was paralysed in the left leg, which was at first rigid below the knee. Philip, aged 4, and Eva Mary, aged 18 months, after a few days of fever were seized with tremors which lasted some days; the youngest child also had strabismus of short duration. The illness with which they were all affected was characterized by pyrexia and headache; there was no rash, nor any sensory changes. There were no epidemics in the village at that time.

Of other epidemics, those recorded from Norway and Sweden are of the greatest importance owing to the pains taken by Wickman and his colleagues to investigate the disease thoroughly from all aspects. In those countries thirteen epidemics in all have been reported, but by far the largest are those investigated by Wickman [15]. He had carefully worked out the details of four epidemics, one of them consisting of over 1,000 cases.

The first occurred in the summer of 1899, and consisted of fifty-four cases; the second of twenty cases in 1903; the third in 1905, numbered 1,031 cases; and in 1906 there was an epidemic of fifty cases. These epidemics all occurred in hot, dry summers, and the number of affected persons reached its maximum in August. The patients were mostly children, but the maximum age-incidence was not so low as in some of the other epidemics; the greatest number were between 3 and 5 years of age, but the disease attacked large numbers of children up to the age of 8. Many adults were, however, attacked, the oldest patient being aged 65, and the mortality was far higher among the elder children and adults than among the younger children. In the large epidemic of 1,031 cases there were 135 deaths, the average mortality of children under 10 years being about 11 per cent., and of the older patients about 26 per cent. Many patients exhibited symptoms of acute polio-encephalitis, either apart from or in association with the ordinary spinal symptoms, and many patients had the constitutional symptoms without subsequent paralysis. To see what light is thrown upon the subject by Wickman's investigations it will be best to consider them under three headings—pathological, etiological, and clinical.

Autopsies were made imperative upon all patients dying during the epidemics, and in this way much valuable material was obtained. All the fatal cases were carefully examined by Harbitz and Scheel [8]. They found that there was a patchy lepto-meningitis more evident on the anterior aspect of the cord, and most severe near areas where the deeper nerve-structures were involved, and that there was great engorgement of the pia in affected areas. The changes in the anterior cornua of affected areas were found to be the same as described by other writers. Harbitz and Scheel, however, regard the lepto-meningitis as the primary lesion, and believe that the inflammatory condition passes in along the vessels, affecting the interstitial tissue and then the large ganglion-cells which derive their nutriment from these vessels. The fact that the inflammation is hæmorrhagic is probably due to the softness of the cord rather than to the severity of the inflammation, as inflammatory changes in any soft organ—as, for instance, the pancreas—tend to be accompanied by hæmorrhage.

As regards the etiology, Wickman proved conclusively that it must be bacterial. He traced several cases where a healthy individual, after visiting an affected centre, returned home, and, having developed the disease himself, infected other people in the previously immune locality. Cases like the above, and also the fact that during the epidemics many patients were affected with paralysis without any febrile symptoms, leave no doubt that acute poliomyelitis (whether developing with febrile onset or coming on without any warning) as a clinical entity is due to some bacterial action. Many different micro-organisms have been separated at different times. Geirswold [5], who investigated the epidemics from the bacteriological point of view, found a poorly staining diplococcus. He could not prove this organism to be the specific organism of the disease, as it was never found alive but undergoing bacteriolysis, and he thought that though a similar organism has not been discovered in any other disease, yet it might be only a coincidence and not the specific organism.

To the clinical knowledge of the disease epidemiology has added much. The first fact of interest is, of course, that it does occur in epidemics, and that these epidemics always occur during the hot summer months. Secondly, that the disease is spread by human beings. The time of year at which all the epidemics have occurred would suggest an analogy with epidemic summer diarrhœa and vomiting, but the way in which Wickman was able to trace the spread from village to village shows that the infection must be born by human beings and is not due to bacterial infection of food

A third point emphasized in the epidemics was the almost universal febrile constitutional symptoms. In many cases paralysis did occur without any constitutional symptoms, but so well recognized were the premonitory symptoms that doctors were able to predict the likelihood of paralysis in a child who only showed symptoms of general malaise.

But perhaps clinically the most interesting feature was the occurrence of many cases which, though not typical poliomyelitis, were obviously other manifestations of the disease which usually attacked the anterior cornua.

Wickman divided the cases into the following clinical types :—

(1) *Spinal poliomyelitis.*—Cases resembling the acute anterior poliomyelitis of text-books. This class was far the largest.

(2) *Ascending myelitis.*—The inflammation rapidly ascending until it affected the basal nuclei; these cases clinically resembled Landry's paralysis.

(3) *Bulbar or pontine cases,* involving the basal nuclei, were generally soon fatal.

(4) *Encephalitis.*—The cerebral cortex was attacked; these cases were identical with superior polio-encephalitis.

(5) *Ataxia.*—A few cases with ataxia resembling Friedreich's ataxia.

(6) *Polyneuritis.*—Many cases, as observed by Gowers [7] and many others, of acute poliomyelitis have symptoms of polyneuritis. This peripheral neuritis causes great local tenderness and appears to be severest in the middle of the course of the nerve.

(7) *Meningitis.*—Many cases of acute poliomyelitis exhibit symptoms of meningeal inflammation. They develop usually before the paralysis and pass off fairly soon. In some cases, however, the symptoms of meningeal irritation were the only symptoms pointing to any spinal lesion.

(8) *The abortive form,* where no paralysis developed, although con-

stitutional symptoms were present exactly resembling those of other patients who were subsequently paralysed. Wickman divides these into three types. (1) General malaise; (2) meningeal irritation; (3) resembling the gastro-intestinal type of influenza.

The occurrence of so many different forms show that though the bacterium or its toxin has a predilection for the anterior cornua, yet it can attack the central and peripheral nervous system at almost any point.

There have been three other large epidemics of late years: one in Queensland in 1903 [10], one in New York in 1907, and one in Melbourne in 1908.

The epidemic in New York [1] [6] [14] was probably the largest that has ever occurred. It was estimated to number several thousands, but unfortunately no careful records were kept. It occurred at the end of a hot, dry summer, and seems in the main to have shown the characteristics of the other large epidemics.

The epidemic in Melbourne was carefully investigated by Douglas Stephens [13]. There were 137 cases and 6 deaths. The summer had been hot and dry, and the largest number of cases occurred towards the end of August. Stephens carried out his investigations as far as he could on the same lines as Wickman.

He considered the disease to be mildly contagious, and suggested that the prominence of gastric or respiratory symptoms might point to different sites of infection. He was, however, unable to prove that point.

In the Melbourne epidemic the cases seem to have assumed the same clinical forms as those described by Wickman. On bacteriological examination a poorly staining diplococcus resembling that described by Geirswald was found. In addition, an organism was isolated resembling the *Staphylococcus pyogenes aureus*; it was not pyogenic, and produced no symptoms on inoculation into the spinal cord of a guinea-pig.

More recently experiments have been carried out by Landsteiner and Popper [9] with material from a case in Vienna. A boy, aged 6, died of acute poliomyelitis within twenty-four hours of its onset. His cord showed the typical histological changes (described by Harbitz and Scheel), but, on staining, no micro-organisms were seen. Cultures taken on agar, serum-agar, and blood-agar were negative. The substance of the cord was inoculated into two rabbits, two guinea-pigs, two mice, and two apes. With the exception of the apes. none developed any symptoms or showed any *post-mortem* lesions. One ape developed typical paralysis after seventeen days and died two days later With the cord of this animal two other apes were inoculated, but without any result. The other monkey was taken very ill six days after inoculation, and died two days later; but, although the cord was much inflamed, there was no typical paralysis.

From these experiments they suggest that the organism is probably too small to be seen.

An epidemic of acute anterior poliomyelitis occurred in the village of Upminster in the summer of 1908. It consisted of eight cases, and lasted from July to September.

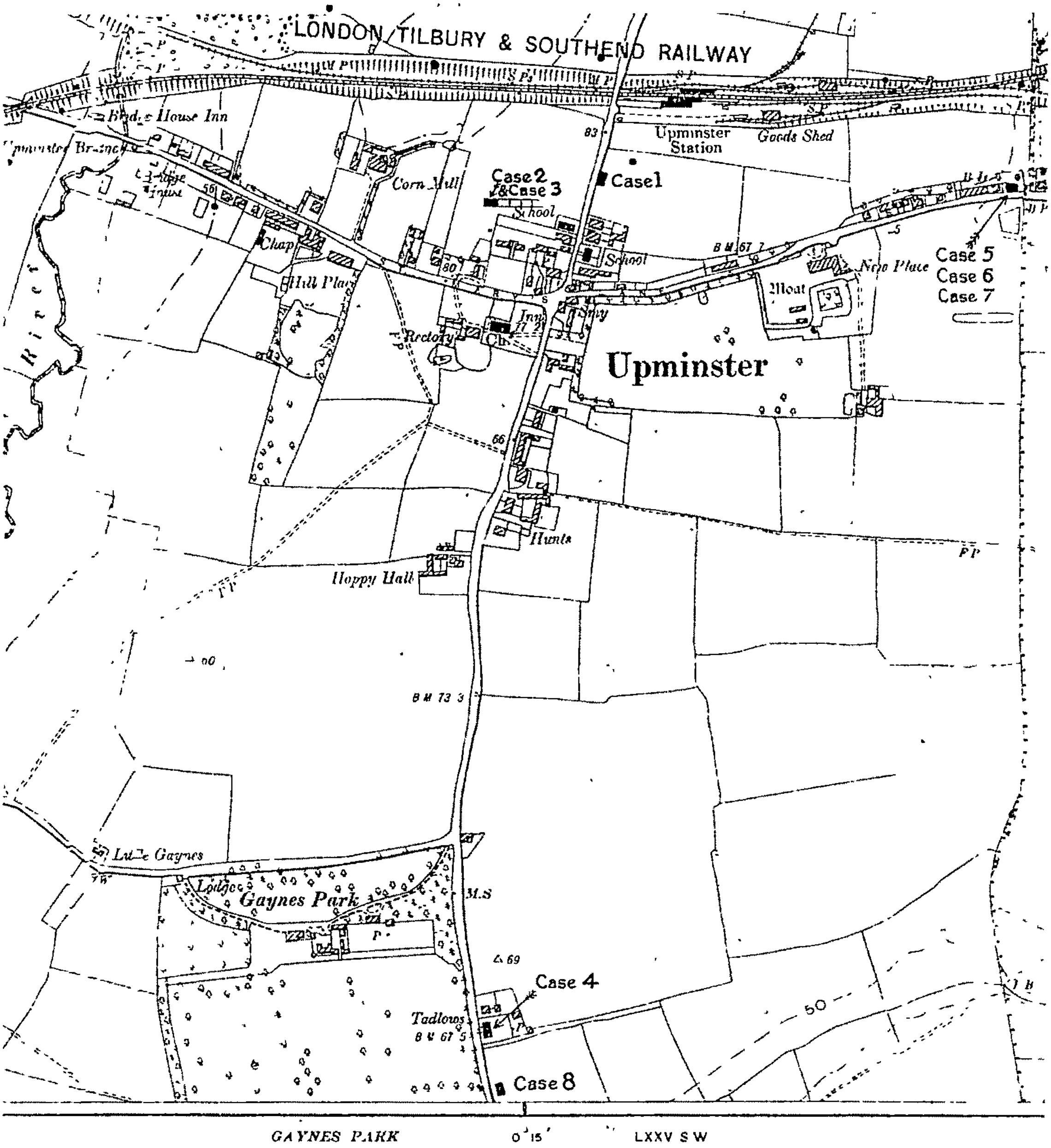
Upminster is a small, straggling village in Essex on a clay soil, with a population of 1,477. It is an agricultural district, and is in no way brought into contact with any country, such as Norway or Sweden, from which infection might have been conveyed. There has never been any similar epidemic in the village, and no case of infantile paralysis has occurred there for several years. The locality has always been considered healthy, and there was no epidemic of any other disease during the year 1908. The months during which the outbreak occurred were hot and dry, but the heat was not excessive.

The first child to be attacked was Gertrude H. (Case I.), aged 8½. She was taken ill on July 27; she was sick, had pains all over her body and a bad headache, and a temperature of 101° F. [38·5° C.]. On August 3 she was found to be paralysed. When examined in February, 1909, she had an almost complete flaccid paralysis of both lower limbs, and was quite unable to stand.

On August 24 three more children were affected: two sisters, Enid (Case III.) and Maud H. (Case IV.)—who, although they bear the same surname as the first patient, Gertrude H., are in no way related to her, and a boy, John C. (Case II.).

John C. (Case II.), aged 13, was taken ill on August 24. He was feverish, his eyes were "glazed," he vomited, and had pain in his lumbar region when moved. He stayed in bed three days, and on August 27, when he got up, fell owing to the weakness of his right leg. The muscles rapidly wasted, and in February, 1909, there was a flaccid paralysis of most muscles of the right thigh and leg.

Enid H. (Case III.), aged 11, and Maud H. (Case IV.), aged 12, had returned to Upminster from Westgate on August 18. On the afternoon of August 24 both sisters were feverish and had pains in the head and stomach. They did not go to bed. The next day they were seen by Dr. Dunlop, and had sore throats and a



Map of the district in which the epidemic occurred. Taken from the Ordnance Survey [6 inches to one mile, or about 1 cm. to 105 metres].

temperature of 101° F. [38·5° C.]. They were sent to bed. Maud got quite well in three days and never developed any paralysis. Enid, between August 28 and 30, had three attacks like severe cramp in the left leg, and on August 30, when she tried to get up, fell, as both legs were weak. When seen in February, 1909, the right leg appeared normal, but the left leg was much wasted and many of the muscles were still paralysed.

The next children to be attacked were three of the M. family. The whole family had been away to Gatestown and returned to Upminster on September 3. The three affected were the middle members of the family, the eldest and youngest remaining well.

The first to fall ill was Stanley (Case V.), aged 8. On September 9 he had a cough, bad headache, was feverish, and had staring eyes. On September 11 it was noticed that he was completely paralysed in his left arm. He rapidly made a partial recovery, but in February, 1909, there was still a flaccid paralysis of many muscles of the left arm. His sister, Eva Rose M. (Case VI.), aged 10, was taken ill September 1[illegible], the day after her brother, with similar symptoms. On September 13 she had lost all power in her neck. Within three weeks she had almost entirely recovered, and in February, 1909, no paralysis was detected.

On September 12 Gladys M. (Case VII.) had a bad headache, vomiting, feverishness, and general malaise. On September 14 she had completely lost the use of both lower extremities. She partially recovered the use of her left leg, and in February, 1909, it appeared quite normal, but there was a flaccid paralysis of most muscles of the right thigh.

The last case was a boy, Basil J., aged 13 (Case VIII.). On September 16 he suddenly became paralysed in his right leg. There was a history of a knock several days previously, but he had been quite well since and the blow was not a severe one, and it seemed justifiable to disregard it. The paralysis came on without any constitutional symptoms. In eight days the leg was quite recovered, and in February, 1909, there was no sign of any paralysis.

Fortunately, though only a small epidemic, it includes all the three clinical types of the large epidemics. Six of the children had constitutional symptoms and then a few days afterwards became paralyzed; one child (Maud H.) had the fever, but developed no paralysis; while Basil J. was paralysed without any constitutional disturbance.

The distribution of the paralysis agrees with the general descriptions, the legs being most commonly affected. In some of the children the eyes seem to have attracted the parents' attention by their peculiar look. but in no case was any definite evidence of polio-encephalitis obtained.

The age of the patients is of interest. All were over 6 years of age, except Gertrude H., who was 3½ years old. About 60 per cent. of all cases of acute anterior poliomyelitis occur in the first three years of life, and, with the exception of Gertrude H., in each family there were younger children who escaped the disease.

As regards the incubation of the disease, the periods of which any information could be gained agree with the figures of Wickman. The child in whom the disease first started had not been out of Upminster, and so the disease must have originated in that village. Enid and Maud H. returned on August 18 and were attacked on August 24; the M. family returned on September 3, and the first was taken ill on September 9: so that in these cases the incubation could not have exceeded six days.

In other epidemics it has not been common for more than one member of a family to be affected, but in this case there were three in one family and two in another. All attempts to trace the means by which the disease spread failed.

Case I.—Aged 3½. Taken ill with pains in the head and vomiting on July 27, 1908. *Paralysis discovered on August* 3. *Examined February*, 1909: *then had flaccid paralysis of both lower extremities and lower part of spine.*

Gertrude J. H. on July 27 fell asleep while out of doors in the evening. She was brought home at 7 o'clock and vomited when she woke up. She was put to bed, but got up next morning. She had severe pains in the head and had to be carried downstairs. On that day she vomited again. The following day, July 29, Dr. Dunlop was called to see her. She had pains in her head and pains all over when she moved. At that time there was no paralysis. On August 3 it was discovered that she was paralysed in both her lower extremities. The paralysis had never been more extensive than it was when she was examined in February, 1909.

She is the youngest of a family of six children. The other children were all at home at the time of her illness, and she was nursed by her two eldest sisters. None of them had any illness at that time. She had not been away from Upminster at any time shortly before her illness.

Condition in February, 1909.—She was a largely-built child, looking about her age. She had always been healthy, and there were no abnormal signs in the heart, lungs, or abdomen. There was obvious wasting of both lower extremities. There was a definite curve in the lumbo-sacral region and wasting of the lower part of the erector spinæ. There were no cutaneous nor vasomotor changes. She could sit up by herself, but was unable to stand. She could extend and flex the toes of her left foot, and could adduct but not abduct the left foot. With the exception of these movements there was complete flaccid paralysis of both lower limbs. The tibialis anticus, the peroneus longus, and the peroneus brevis of the left foot appeared to be unaffected. The extensors and flexors of the toes and foot on that side were working, but appeared to be weak. With the exception of these muscles there were great wasting and loss of tone of all the muscles of both lower extremities. The glutei on both sides were affected, and also the lower part of the erector spinæ. As the child was frightened the electrical reactions could not be tested. She was having electrical treatment, however, and the operator stated that the muscles did not react to faradism, but gave a slow contraction when stimulated by the galvanic current. The knee-jerks on both sides were absent. The plantar reflex on the left side was flexor; on the right side it was absent. Sensation appeared to be unaffected. The sphincters were unaffected.

Case II.—Aged 13. *Taken ill August* 24, 1908, *with headache and vomiting. Stayed in bed for three days. August* 27, *when he got up, his right leg was weak. Examined February*, 1909: *flaccid paralysis of right leg and thigh.*

John C. was taken ill on August 24, 1908. His head ached and he had pain in the lumbar region when he moved. He was feverish, and he vomited several times. His parents say that his eyes were "glazed." He stayed in bed for three days, and at the end of that time he felt quite well. On August 27, when he got out of bed, his right leg was weak, so that, although he could walk without assistance, he was liable to fall down. He had no pain in his leg and there was nothing peculiar to be seen. The muscles rapidly wasted both above and below the knee. He attended the London Hospital until September 26, and was treated with electricity and massage. After that date the treatment was continued at home.

He is considered the healthiest of a family of nine children, five of whom are older and three younger. The rest of the family were at home, but none of them were attacked by any illness.

Condition in February, 1909.—He was a well-grown, strong, healthy-looking boy, who had never had any serious illness before. Three years ago he was hit on the vault of his cranium by a piece of lead piping; the bone was not affected, but the scar remains, and he has occasional headaches since, but no other trouble. There were no abnormal signs in any of the abdominal or thoracic viscera. There was obvious wasting of the whole of the right thigh and leg, especially in the region of the quadriceps and psoas. The foot was in a position of slight equinus, with hyper-extension of the big toe. There were no cutaneous nor vasomotor changes. On measurement, the right leg was found to be considerably smaller than the left, both above and below the knee.

Below patella	*Right*	*Left*
10 cm.	25·7 cm.	26·4 cm.
15 ,,	28·3 ,,	29·6 ,,

Above patella	*Right*	*Left*
8 cm.	28·5 cm.	31·5 cm.
12 „	31·3 „	35·6 „
16 „	33·2 „	37·4 „
24 „	34·5 „	39·4 „

He could walk quite well without assistance. There was slight dropping of the right foot and updrawing of the big toe. In the right leg all the muscles except the tibialis anticus were working, but they were not so strong as in the left leg, and the extensors were more affected than the flexors.

In the thigh he could not fully flex the thigh on the abdomen, nor could he fully extend the leg. Flexion, external rotation, and abduction were good; adduction and internal rotation were weak. The psoas and quadriceps in the right thigh and the tibialis anticus in the right leg were the only muscles in which there was no action. All the muscles of the thigh, except the quadriceps and psoas, reacted well to the faradic current The sartorius and glutei reacted well.

In the leg all muscles but the tibialis anticus reacted well to the faradic current. The tibialis anticus gave a slow action to 4 ma. of galvanic current, the contraction being greater on closure of the current when stimulated by the anode than by the kathode No reaction was obtained to the constant current in the quadriceps or psoas with 4 ma The knee-jerk was not obtained in the right leg, the plantar reflex was flexor. There were no sensory changes.

Case III.—Aged 11. *Illness began August* 24, 1908, *with headache and feverishness August* 25: *went to bed, and during next five days had attacks resembling cramp in her left leg. On August* 30 *it was discovered that she was paralysed in both her legs. Examined February,* 1909. *flaccid paralysis of some muscles of left leg, but no muscle of right thigh or leg obviously affected Sister of Case IV, but no relation to Case I*

Enid H. had been staying at Westgate and returned to Upminster on August 18. On August 24, in the afternoon, she had a bad headache and was feverish, but did not go to bed. On the evening of the next day she went to see Dr. Dunlop. She then had a headache, slight sore throat, and a temperature of 101 F [38·5° C.], but no paralysis. She was sent to bed. Between that time (August 25) and August 30 she had three attacks like bad cramp in the left leg. These attacks lasted about one minute, and were so severe that they made the patient scream. On August 30 she felt quite well and tried to get up, but fell She was again examined by Dr. Dunlop The sore throat had recovered and the constitutional symptoms had subsided. The right leg was weak but the knee-jerk was present. The left leg was paralysed, and the knee-jerk was absent

The right leg gradually got better. The muscles of the left leg wasted rapidly, but after a few weeks she began to regain power and could walk about.

She is the second child of a family of four children. All the children had been away together at Westgate and they all returned to Upminster on August 18 Her elder sister (Case IV.) was taken ill on the same afternoon with pains in the head and stomach, but she did not develop any paralysis. A younger sister, aged 5, and a brother, aged 18 months, were not affected.

Condition in February, 1909.—A good-looking girl, looking more than her age. She has always been healthy and has had no serious illness. There were no abnormal signs in the heart, lungs, or abdomen. The right leg was well developed and was not affected in any way. In the left leg there was obvious wasting both above and below the knee, the wasting being greatest on the front of the thigh. There were no cutaneous nor vasomotor changes. The circumference of the left leg at various levels was considerably less than that of the right leg.

Below patella	*Left*	*Right*
10 cm.	24·0 cm.	26·25 cm.
15 ,,	20·5 ,,	23·5 ,,
Above patella		
8 cm.	26·0 ,,	30·0 ,,
16 ,,	28·25 ,,	35·5 ,,
24 ,,	30·0 ,,	38·25 ,,

She could walk well, though she dragged the left leg slightly. The movements of the right leg and thigh were perfect. She could not extend the left knee to any material degree, and all other movements were weak. In the left thigh the quadriceps (including both vasti) were not working, the hamstrings, the abductors, and adductors were working but were weak. The glutei on both sides were perfect The muscles below the knee on the left side were working, but were weak compared with the right side. All the muscles of both extremities reacted well to the faradic current, except the left quadriceps. The left quadriceps did not react to a galvanic current of 4 ma.

The knee-jerk was present on the right side, and absent on the left. The plantar reflex was flexor on both sides.

There were no sensory changes.

Case IV.—Aged 12, *sister of Case III., no relation to Case I. Taken ill on August* 24 *with pains in stomach and head In bed three days. Never any nervous symptoms*

Maud H. returned from Westgate on August 18 with her sister Enid (Case III.) and the rest of the family. On August 24 she was taken ill with headache and pains in the stomach. Her illness started rather later in the day than her sister's, and it was not so severe. She had no sore throat and no pains in her limbs. On August 25 she went to bed, as her sister had been ordered to do so She stayed in bed for three days, and at the end of that time she felt quite well and was allowed to get up. She had no paralysis or muscular weakness.

Condition in February, 1909.—Not so strong or well-developed a girl as her sister Enid (Case III.), but quite healthy. There were no abnormal signs nor weakness in any organ or in any limbs.

Case V.—Aged 8. September 9 had a cough, a bad headache, and was feverish. September 11: was found to be entirely paralysed in his left arm. Improved greatly but not completely. When examined in February, 1909, had wasting and paralysis of many muscles of his left arm. He is the brother of Cases VI. and VII.

Stanley M. had been away with the rest of the family to Gatestown, in Essex, and had returned to Upminster on September 3. On September 9 he had a cough, a bad headache, and "glaring" eyes. He was feverish and had lost his appetite. He was put to bed. On September 11 he did not use his left hand to feed himself, and it was found that he had lost all power in his left arm. In a few days he could lift his left arm almost to a right angle, but could not bend it nor grasp anything in his left hand. The muscles rapidly wasted. There was no rash nor vomiting, no pain, and no loss of power elsewhere. He attended the London Hospital for electrical treatment, and his condition much improved.

Stanley M. is the third child of a family of five. The whole family had been away together and returned on the same day. The eldest child, aged 14, and the youngest, aged 12 months, remained quite well. The other two, Eva Rose (Case VI.), aged 11, and Gladys (Case VII.), aged 6, were affected with constitutional symptoms, like those of Stanley, two and three days later respectively, and each subsequently developed a flaccid paralysis, but in different parts of the body.

Condition in February, 1909.—He was a healthy-looking boy, but not largely built. He had diphtheria in 1907, but has always been bright and healthy. The left hand could be held in any position, but was usually held fully pronated, the wrist dropped and the proximal phalanges hyper-extended The first phalanges of the ring and little fingers were extended; the second and third were slightly flexed. The thumb was held extended and abducted. The muscles of the whole limb were wasted and had lost their tone, those of the hand being most affected, and those around the scapular least. There were no cutaneous or vasomotor changes.

On measurement the left arm and forearm proved to be definitely smaller than the right.

From the tubercle of the acromion	*Right*	*Left*
5·0 cm.	19·0 cm.	16·5 cm.
10·0 ,,	17·8 ,,	15·6 ,,
12·5 ,,	17·5 ,,	15·6 ,,
From external condyle		
5·0 cm.	17·8 ,,	15·6 ,,
7·5 ,,	16·5 ,,	13·6 ,,
10·0 ,,	14·3 ,,	12·0 ,,

He could perform any movement with his left arm or forearm, though less powerfully than with his right; but in the left hand there was no movement of the small muscles of the thumb, of the interossei, or of the little finger.

The left trapezius was not affected. The deltoid, rhomboid, supraspinatus, infraspinatus, and pectorals were all slightly affected. All the muscles of the upper and lower arm were working but not strongly, especially the flexors of the upper arm and the extensors of the forearm. Below the wrist none of the small muscles were acting at all.

All the muscles of the arm and forearm reacted well to faradism, but none of the small muscles of the hand responded to this stimulus. The small muscles of the thenar and hypothenar eminences reacted well to 5 ma. of galvanic current; the interossei also reacted to the same strength of current, but more slowly. In all cases, however, the contraction caused on closing the current was greater when stimulated by the kathode than by the anode.

The reflexes were obtained and were normal.

Sensation was not affected.

Case VI.—Aged 10, *sister of Cases V. and VII. September* 10, 1908, *had headache, dry, troublesome cough, and pain in suboccipital region when moved. September* 13 *had lost all power in her neck. Partially recovered in ten days, but was three weeks before could hold her head up for any length of time. Examined February*, 1909, *and nothing abnormal found.*

Eva Rose M. had been away with the rest of the family to Gatestown and had returned to Upminster on September 3. On September 10, one day after her brother Stanley (Case V.) had been taken ill, she had a bad headache, a dry, troublesome cough, with hot sweats and severe pain in the suboccipital region on movement. She did not vomit and had no rash. On September 13 she had lost all power in her neck. There was no wasting and both sides appeared to be affected equally. In ten days she had partially recovered, but for about three weeks could not hold up her head for long at a time.

Condition in February, 1909.—Like the rest of the family, she is rather small for her age. She had had the usual infantile complaints, but since five years of age has been quite well. She said that at times (for instance, when cold) her neck felt rather weak, but there was nothing abnormal to be seen or detected either in her neck or in any other part of her body.

Case VII.—Aged 6, *sister of Cases V. and VI. September* 12, 1908, *had a bad headache, vomiting, feverishness, and general malaise. September* 14 *had entirely lost the use of both lower extremities. Gradually recovered the use of the left leg, but the right never quite recovered. When examined in February*, 1909, *the left leg was not obviously affected, but there was flaccid paralysis of most muscles of the right thigh.*

Family History—Gladys was the third of the M. family to be taken ill, two days after her sister Eva Rose (Case VI.) and three days after her brother Stanley (Case V.). The eldest and youngest were never affected, though exposed apparently to equal risk of infection.

Condition in February, 1909.—A bright, plump child of moderate size for her age, and a good colour. She has had no previous illnesses. The muscles of the right thigh were obviously wasted and lacked tone. The muscles of the right leg and of the left thigh and leg appeared perfectly normal.

There were no cutaneous or vasomotor changes.

Below the patella both legs were equal; above the patella the right leg was the smaller.

Above patella	*Right*	*Left*
5·0 cm	23·8 cm.	26·6 cm.
7·5 „	25·4 „	29·3 „
10·0 „	27·0 „	30·5 „

She walked and ran without any limp, difficulty, or unsteadiness. She could perform any movement, but there was not so much power in the right thigh as in the left.

The right quadriceps was very weak, the rectus and vastus externus not acting at all. The adductors and hamstring acted moderately, the biceps appearing to be the most affected of the hamstrings. All other muscles of both lower extremities acted well.

The right rectus did not react at all to the faradic current; the right vastus internus reacted badly. All the other muscles reacted well to the faradic current. All the muscles, including the rectus, reacted normally to a galvanic current of 5 ma.

The knee-jerk was stronger on the left side, but was present on the right. On both sides the plantar reflex was flexor

There were no sensory changes.

Case VIII.—Aged 13, *without any constitutional symptoms, was paralysed in his right leg on September* 16, 1908. *In eight days was quite well. Examined February*, 1909, *and nothing abnormal found.*

Basil J. had been away at Bexhill during August and had returned to Upminster on August 31. On September 10 he was kicked in the lumbar region by a man. The kick was not severe. He went to bed quite well, and went to school next day. On the following morning, September 12, he fell down when he got out of bed, owing to weakness of his right leg. He was examined by Dr. Dunlop. He was in no way ill; he had no cough, no sore throat and no pain. There was no knee-jerk in the right leg; the muscles were flaccid, and there was no movement below the knee. There was no bruising of the spine. After three or four days he began to improve, and in eight days was quite well.

He has an older and a younger brother, both of whom were at home at the time of his illness, but they remained quite well.

Condition in February, 1909.—He was a fine, muscular boy in excellent health, and he had never had any serious illness. There was no weakness nor loss of tone of the right leg. Measurements showed the right leg to be slightly larger than the left. The knee-jerks were equal on both sides, and there were no vasomotor changes.

REFERENCES.

[1] Berg, H. "Poliomyelitis Anterior as an Epidemic Disease," *Med. Rec.*, New York, 1908, vol. lxxiii., pp. 1-6.

[2] Buzzard, Farquhar "On Certain Acute Infective or Toxic Conditions of the Nervous System," *Brain*, 1907, vol. xxx., p. 1.

[3] Buzzard, T. "Cases Illustrating the Infective Origin of Infantile Paralysis," *Lancet*, March, 1898, vol. i., pp. 847-850.

[4] Caverly, C. S. "History of an Epidemic of Acute Nervous Disease of Unusual Type," *Med. Rec*, New York, 1894, vol. xlvi., pp. 673-677.

[5] Geirswold, M. "Epidesmisk poliomyelit, bacteriologiske undersogelser," *Norsk. Mag. f. Laegevidensk*, Kristiania, 1905, vol. iii., pp. 1280-1300.

[6] Gibney, V., and Carleton, Wallace. "The Recent Epidemic of Poliomyelitis," *Med. Rec.*, New York, 1907, vol. lxxii., p. 873; "The Epidemic of Infantile Paralysis," *Med. Rec*, New York, 1907, vol. lxxii., p 695.

[7] Gowers, Sir W. "A Case of Anterior Poliomyelitis and Multiple Neuritis," *Clin. Soc. Trans.*, 1891, vol. xxiv., pp. 127-133.

[8] Harbitz, F., and Scheel, O. "Pathologisk-anatomisk undersogelser over tilfaelde af akut poliomyelit og beslegtede sygdomme fra epidemierne i Norge, 1903-6," *Norsk. Mag. f. Laegevidensk*, Kristiania, 1907, vol. v., pp. 1045-1168. (Translation in *Journ. of Amer. Med.*, Chicago, vol. xlix., pp. 1420-1425.)

[9] Landsteiner and Popper. "Uebertragung der Poliomyelitis acuta auf Affen," *Zeitschr. f. Immunitats-forschung* (I. Thiel), Orig., 1909, Bd. ii., Heft iv., S. 377.

[10] McPherson "Epidemic of Infantile Paralysis," *Australasian Med. Gaz.*, 1904, vol xxiii., p. 426.

[11] Medin, O. "En epidemi af infantil paralysis," *Hygeia*, Stockholm, 1890, vol lii.

[12] Pasteur, W "An Epidemic of Infantile Paralysis occurring in Children of the same Family," *Clin. Soc. Trans.*, 1897, vol. xxx., pp 143-150

[13] Stephens, H. Douglas. "Summary of an Epidemic of 135 Cases of Acute Anterior Poliomyelitis occurring in Victoria in 1908," *Intercolon. Med. Journ. of Australasia*, 1908, vol. xiii., pp. 573-582.

[14] Terriberry, G. F. "Some Observations on the Recent Epidemic of Acute Poliomyelitis in New York City," *Med. Rec.*, New York, 1907, vol. lxxii., p. 921.

[15] Wickman, I. "Beitrage zur Kenntnis der Heine-Medinschen Krankheit," Berlin, 1907.

Publications Recently Received.

[Notes on a book under this heading do not preclude a subsequent review.]

Syphilis and Nervensystem. (Neunzehn Vorlesungen für praktische Aerzte, Neurologen und Syphilidologen.) Von Dr. Max Nonne. Zweite vermehrte und erweiterte Auflage. Mit 97 Abbildungen im Text. S. 699. Berlin: Karger, 1909.

Syphilis (as manifested by its effects on the nervous system) is here treated in the fullest manner. Each lecture forms a chapter dealing with one aspect of the subject, illustrated with admirable clinical cases. Professer Nonne first deals with the nature of syphilis in the light of modern research, and then gives a full description of the anatomical appearances produced by the disease in the nervous system. He then passes on to arteritis and its effects, to meningitis of the superior surface of the brain and of the base, including lesions of the cranial nerves. The neuroses and psychoses of syphilis are described and are distinguished from dementia paralytica. Three lectures are devoted to various conditions of the spinal cord and its membranes due to syphilis. Tabes and its relation to syphilis is fully considered, and this is followed by an interesting account of cerebrospinal syphilis. Chapters on hereditary syphilitic nervous disease, on treatment, on cyto-diagnosis, and on Wassermann's reaction complete this most valuable work. Its admirable arrangement and its completeness make it an ideal reference book for the practical neurologist.

Klinische Beiträge zur Lehre von der Hysterie nach Beobachtungen aus dem Nordwesten Russlands. Von Dr. Georg Voss. S. 300. Jena: Fischer, 1909.

The author studied 123 cases of hysteria which came to the Marien hospital in Petersburg, and on this material has based a useful clinical monograph. Chapters are devoted to alterations in sensibility, the reflexes, the special senses, vasomotor and trophic changes, and affections of motility. The work of others is invariably considered, and the views of the author are in accord with those of most neurologists. Thus he does not believe that the sensory disturbances and the limitation of the visual field are phenomena suggested by medical examination, and looks upon them as of diagnostic importance.

The Criminal Responsibility of Lunatics: a Study in Comparative Law. By HEINRICH OPPENHEIMER, LL.D., M.D. Pp. 275. London: Sweet and Maxwell, 1909.

This work is an attempt to bridge the gulf between legal and medical opinion on criminal responsibility by one who is both a lawyer and a medical man. Chapters are devoted not only to the English law, but to a careful survey of the laws of other countries, and the author describes the actual working of the paragraphs on criminal responsibility in the various European States The "knowledge test," freedom of will, and the power of self-control are fully dealt with The treatment of moral insanity, partial insanity, and "the critical moment" is less complete, probably because they require wide clinical knowledge and familiarity with actual examples of the conditions described. This book is an interesting attempt to deal with a subject rendered difficult by the fundamental difference in outlook of lawyers and medical men.

Attention. By W. B. PILLSBURY. Pp. 346. London: Swan Sonnenschein, 1908.

This work formed originally one of the volumes of "The Bibliothèque Internationale de Psychologie expérimentale," and the present volume is a republication with numerous additions. It contains an account of all the recent methods for studying attention and the results obtained by their use. Attention is treated physiologically, psychologically, and philosophically. Chapters are devoted to the relation of attention to ideas, perception, memory, will, reason, and feeling. Every chapter concludes with a useful summary, and the book forms a valuable record of our present knowledge of this difficult subject.

Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete der Neurologie und Psychiatrie. XII. Jahrgang: Bericht über das Jahr, 1908. S. 1312. Berlin: G. Karger, 1909.

This volume retains all the excellences of previous numbers. No working neurologist can afford to be without this work of reference on his shelves.

A Text-book of Mental Disease. By EUGENIO TANZI. Authorized translation from the Italian by W. FORD ROBERTSON, M.D., C.M., and T. C MACKENZIE, M.D., F.R.C.P. Pp. 803. London: Rebman, 1909.

This is a well-arranged text-book on clinical lines. The illustrations are good and the translation excellent.

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EDITOR.

[March, 1910.]

BRAIN.

PART IV., 1909.

Original Articles and Clinical Cases.

THE HUGHLINGS-JACKSON LECTURE ON SPECIAL SENSE DISCHARGES FROM ORGANIC DISEASE.

BY SIR WILLIAM R. GOWERS, M.D., F.R.S.

Mr. President and Gentlemen,—To deliver the Hughlings-Jackson Lecture is a task equally pleasant and difficult. Its pleasure and its difficulty arise from the same source. The object of the lecture is to honour the distinguished worker who is still with us, by an attempt to promote the researches which have thrown such bright lustre on his name, and, if it be possible, to carry a step further the results he has reached, or at least to provide the means that may enable others to achieve this result. But to make such an effort is to realize its difficulty. Knowledge grows most slowly, for the essential facts are rare, and those already gathered have been made to yield their lessons by the Master himself, beyond whom we cannot hope to go. I fear, therefore, my attempt will involve disappointment to you and to myself.

The subject to which I propose to limit my consideration is the symptoms of epilepsy which are related to the so-called special senses—those of smell, vision, hearing, taste, and perhaps touch—and especially the indications of such epileptic symptoms as are occasionally presented by organic disease. Examples of such epileptic discharges from organic disease, demonstrable in nature and seat, are rare. It is important to lose no opportunity of examining carefully such as are met with.

Olfactory.

Anatomically, the special senses begin with that of smell, and the olfactory discharges were the first to receive special study from

Hughlings Jackson, whose analytical observation has exerted a fascinating and inspiring influence on workers in every country. He published a paper, calling attention to their complex and instructive peculiarities, as long ago as 1866, a paper that was referred to, and quoted, by Sander, of Berlin, in an account of a fatal case published in 1874, when he also considered the few cases he had been able to find recorded by previous writers, one in 1824 unintelligible to those who do not know the place indicated by the "middle of the hemisphere." Hughlings Jackson returned to the subject again and again, in penetrating discussions of individual cases, especially in 1899 [3] in a paper in which Purves Stewart described six fatal cases that had been published, by Jackson and Beevor, Jackson and Colman, Anderson, Nettleship, Sander and McLane Hamilton.

Another case was published by C. K. Mills in 1908 [6], who also added two others described by Linde [4] and by Southard [7], raising the number to nine. Two cases have also been published by Buzzard in 1906 [1]; although in one of these there was no post mortem, the nature and seat of the disease were ascertained by surgery, and it is therefore deserving of inclusion. I have two other fatal cases to describe to you to-night—indeed, four—and I find that Sander, in addition to his own case, quoted others, of which two are important—one by Lockemann [5] (in 1861), and another by Westphal [10] (in 1863).

The case described by Buzzard was a girl, aged 21, who was found to have a tumour in and about the right hippocampal gyrus, which had destroyed the uncus and had filled the descending cornu of the lateral ventricle. It had given rise to a prolonged sensation of a bad taste in the mouth and to a sound of bells in the head, neither associated with her epileptic attacks.

Buzzard also mentioned a case in which the lesion was revealed by surgery, a man who had at first a frequent subjective sound like the loud clang of a piano, accompanied, after a time, by a very unpleasant smell and a keen feeling of reminiscence. A glioma was found infiltrating the base of the right temporal lobe, but the operation for its removal was not long survived, and no *post-mortem* examination was allowed.

Lockemann's case was that of a woman, aged 55, who died from a malignant tumour of the brain which extended from the left frontal lobe to the olfactory region, and caused, for a time, a peculiar olfactory aura of frequent attack; the sensation was described as variable and not unpleasant.

That of Westphal was a syphilitic nodule in the pia mater beneath the frontal lobe, which had destroyed the right olfactory lobule and caused a very offensive smell as the warning of epileptic fits.

The case I have specially to describe is that of a man, aged 37, who was admitted to the National Hospital in January, 1905, having suffered for three and a half years from frequent slight attacks, of which several occurred daily, often four or five. In these there was, first, a smell and taste of gas; he could not compare it to any known odour, but it seemed to be something like sulphuretted hydrogen. It caused salivation, and eructation from the stomach. The flow of saliva was so abundant that it would run from the mouth, and often filled a tea-cup. After the sense of smell he had a slight sense of turning, and if he walked he had a tendency to walk fast, almost to run. Each attack lasted from one to three minutes. Consciousness was not lost. He occasionally had a more prolonged sense of the same odour.

Six months after these commenced he had a fit with loss of consciousness, and the onset was marked by sleepiness, but with no sensation of smell or of taste. During the next year he had seven others of the same kind. There was apparently general convulsion, since in some of these fits his tongue was bitten. The attacks with smell continued

Five months before admission he had severe headache for a week, commencing on the right side of the back of the head and neck, and spreading forwards over the right temporal region to the forehead and felt over both eyes. Afterwards he had another briefer attack, but on the whole he had very little headache. Immediately after the first he had diplopia for two or three weeks, and he noticed that his eyesight became poorer, objects being indistinct, especially to the left. For about three months he had noticed that he could not smell so well as formerly.

He was a well-developed man, and his mental state presented no abnormality. His smell was defective, especially on the right side, but did not seem to be lost. There was almost complete left hemaniopia, the loss extending almost up to the middle line, but it spared the region of the fixing point. The right half field was restricted in the upper periphery, especially in the right eye. The left optic disc presented considerable papillitis about four discs breadth in width, but with only slight swelling (two diopters, Mr. Gunn). It was grey in tint, with a few hæmorrhages on each side. The right optic disc appeared normal. The only morbid appearance it presented was a little capillary redness in the upper part.

There was considerable loss of power in the left external rectus, and in the right internal rectus on convergence, but alone it acted well. There was no nystagmus. The conjunctival and corneal reflexes were diminished on the left side, but there was no defect of sensation on the face or weakness of the masseter. Slight weakness of the face on the left side existed in all parts. Hearing was good on each side. The tongue and throat presented no symptoms. The strength of the left hand seemed a little diminished. Both knee-jerks were brisk, but there was no foot clonus. The left abdominal reflex was slighter than the right.

He had only occasional headache—not severe. There was marked tenderness to firm pressure on the right side at the temporal and lower part of the parietal region. No sound could be heard on auscultation there.

In February he was trephined by Sir Victor Horsley. The right parietal and temporal regions were exposed. The brain was bulging, but no tumour could be seen on the surface, or on raising the temporal lobe; but this was firmly adherent to the dura mater near the tip. An oblique incision into the substance of the lobe revealed an infiltrating growth near the anterior extremity, which could only be partially removed, in pieces. Hæmorrhage occurred from many vessels which had to be tied. The operation was borne badly, and the patient died a few hours after its completion.

At the necropsy it was impossible to discern the precise size and position of the growth in the temporal lobe. It was found to have compressed the chiasma and right optic tract, and it was continuous with a mass of similar growth in the white substance of the frontal lobe, 1¾ in. in diameter. The highest part of this was ½ in. below the second frontal gyrus, and it had reached the orbital surface to the outer side of the middle The growth was a glioma.

I shall have presently to describe another case in which the fits were preceded by an auditory warning at first, and subsequently by an olfactory aura, consequent on the descent of a growth towards the apex of the temporal lobe.

Twelve of the cases cited above were so extensive as to have no precise localizing value; of these, eleven were of tumours and one of meningitis. Another, that of Westphal, had apparently produced the symptom by destruction of the olfactory bulb and tract. There was extensive meningitis in the case of Southard, but there was also an aneurism which had destroyed the uncus. In only one case was there an isolated lesion of this structure—a small spot of softening within the uncus—and in this case, described by Hughings Jackson and Colman, there were no sensations of smell, only the movement of the mouth and jaws which is so suggestive of a sensation of taste. This was present also in the case of Southard, in which the uncus was destroyed by an aneurism. Although the symptom is probably a distant secondary effect, and is present in many cases in which there is no evidence of disease in this region, we are almost compelled to regard it as evidence of a gustatory sensation. We must remember, moreover, that Jackson, in proposing the name "uncinate epilepsy," for these attacks with an olfactory or gustatory aura, expressly disclaimed suggesting more, as the seat of disease, than the general region traversed by the olfactory roots. Where these fibres end, in the various structures they penetrate, remains to be discovered.

A putrid odour was occasionally described. Regarding this, caution is necessary when it does not attend other distinct epileptic symptoms. It may be an isolated manifestation of suppuration in one of the nasal sinuses.

Several peculiar features frequently attend these cases of epileptic attacks with an aura of smell, but little can be added to the instructive discussions they have received from Hughlings Jackson. I may mention that he has called attention to the observations of W. G. Spencer [8] on the arrest of respiration, at the end of inspiration, produced by stimulation of the anterior perforated spot, over which passes the outer root of the olfactory tract, and into which some of its fibres pass, as, perhaps, explaining the sense of dyspnœa occasionally associated with an olfactory aura. We are reminded of it by some cases of apparently idiopathic epilepsy. For instance, in one a sense of dyspnœa seemed to have a nasal origin. The first thing was a stifling sensation in the nose, as if the patient could hardly breathe, followed by a sudden sense of a peculiar momentary bad smell.

The psychical condition which often attends the slighter attacks in cases in which there is an olfactory sensation has been termed the "dreamy" state. There seems to be only a partial impairment of consciousness, or the loss of this occurs so slowly that a distinct recollection is retained of the transient sensations which precede it. The term "dreamy" is often spontaneously applied to the memory of other slight attacks, especially of those in which some definite sensations were experienced. So also with the impression of recurrence, or of precurrence, it might be more accurately termed—the clear belief that the same feeling has been experienced before. This is a phenomenon of normal brain states, or presumably normal, as Oliver Wendell Holmes has so well impressed upon us in his "Autocrat of the Breakfast Table," in connection with Day and Martin's blacking. Much more strange, and at present beyond our power of conceiving an explanation, is the sense of dread, intense and causeless, which has been marked in several cases.

In ordinary epilepsy we sometimes meet with a slighter sensation of smell associated with other definite and deliberate sensory perceptions. In one case the sound of bells, with red and blue lights, was followed by a distinct putrid odour. Another patient always had a peculiarly complex aura. A sensation in the left hypochondriac region seemed to ascend the left side of the thorax in a jerky motion, and, at the clavicle, it became a sound, and could be heard as well as felt; when it reached the level of the ear it was like the hissing of a railway engine. Then he suddenly saw an old woman in a brown dress, who offered him something that had the smell of Tonquin beans. The woman then disappeared, and two round lights appeared before him, which got nearer and nearer with a jerky motion, and he lost consciousness. The warning was always the same in every detail. Such cases are, doubtless, due to a secondary involvement of an olfactory centre in a discharge beginning elsewhere and spreading widely.

AUDITORY.

Organic disease sometimes causes attacks in which the warning is a subjective sound. As might be expected, the cause is generally a tumour of the upper part of the temporal lobe, often subcortical, beneath the first temporal in which experiment indicates the situation of the auditory centre. But it will be remembered that experimental extirpation of this has never been followed by permanent loss of hearing, neither has this result followed enduring destruction of this region by disease in man.

The effect of a tumour in the region was shown by the case recorded by Buzzard which I have already mentioned, in which a glioma of the base of the temporal lobe caused, at first, sudden sounds like the twang on a piano-chord, and, later, sensations of smell. This effect is not rarely met with, and it is noteworthy that the discharge of the centre is often due to a growth in the white fibres below it. It was so in a remarkable case which came under my notice many years ago at the National Hospital, where the patient was under the care of Dr. Hughlings Jackson. The details of the case have never been published, and Dr. Jackson, with characteristic kindness, has placed them at my disposal this evening. The notes are long, for they comprehend the patient's four years' history, but I will abridge them as much as I may, and I beg you to accept my regret if they weary you; they are important.

The patient was a man of 39, admitted in November, 1875. He was a painter, but without sign of lead poisoning. There was no history of syphilis. He was left-handed, but had been taught to write with the right hand. For two years he had suffered from fits; before then he was well. He had also suffered much from occipital pain, increased by a cough. There was no paralysis. The fits had occurred about once a week during the two years. For the first six months there was no warning; subsequently there was an auditory aura. The slighter attacks began by a humming sound in the left ear; those of greater severity, by a loud sound like a peal of bells, also to the left. The fits continued after admission. The duration of the loud sound was about three minutes, and it ceased before the convulsion began. Just before it ended, the patient felt a burning sensation in the left external auditory meatus and concha, like the contact of hot coal. The burning sensation then passed down the left side of the neck, and round the back of the neck to the right ear, and also across the forehead. In the neck, and indeed in each part to which it passed, it was accompanied by a sensation that the part was fixed and could not be moved. The burning then passed down the thorax, front and back, to the leg and foot, where it became a numbness. After the leg the sensation passed down the arm to the hand, which felt heavy and fixed. From the onset there was intense frontal pain. The head shook rapidly from side to side, and the eyelids were quivering. Sparks also appeared before the eyes, always yellow, deep yellow at first, afterwards lighter; with the onset of the burning heat, the face became drawn towards the left by tonic spasm, and afterwards there was trembling of the limbs of both sides. If he could get anyone to squeeze his head, the sensation lessened, and he did not lose consciousness, otherwise he became unconscious and his tongue had often been bitten, but no severe convulsion was seen in the hospital. In other attacks of the same character the arm was affected by the sensation before the leg, and his description was that the arm seemed as if lying in a bed of stinging nettles. The attacks were always followed by intense headache. When admitted the optic discs were normal, but in three months optic neuritis slowly developed. Soon afterwards left-sided hemianopia was found, the loss almost reaching to the medial line. The fits he had in the hospital were less frequent. Each was followed by transient weakness of the left side, the only paralytic symptom. His headache improved. After six months he went out, for the same time, and returned at the end of October, 1876. The fits had continued; many had occurred without warning, but those which he had after re-admission presented the same aura, the sound of a peal of bells, now followed by pain at the back of the left hand, which passed up the arm to a little above the elbow, and was then felt in the left side of the face. Tingling followed in the left leg, which passed up the thigh; then he fell, unconscious. The arm was the seat of constant tremor, which was present also in the leg when he was sitting, but ceased when he stood. It began suddenly in June in a violent form, soon after rising. In the arm it was coarser than in paralysis agitans, and involved all the joints, but was greater in the wrist and fingers than in the elbow or shoulder. A movement stilled it for a time, but it soon returned. The left arm had become distinctly weak; the grasp was 35 with the right hand, but only 10 with the left. The leg seemed strong. The optic neuritis was subsiding, but vision was considerably impaired.

He did not then stay long in the hospital, and the notes are brief, but he was again admitted in October, 1878. Continuous occipital headache still troubled him. Left hemiplegia had gradually developed, and had attained a considerable degree. The neuritis had led to consecutive atrophy with still more impairment of sight, greater in the right eye. The left hemianopia persisted. Hearing was noted to be good on each side; smell also was good and equal; taste seemed diminished. His fits had become less frequent, and many appeared to have been bilateral. He still had frequent severe occipital headache

Many fits occurred after he returned to the hospital; the description of them in the notes now never mentions an auditory aura. In those that were severe, the convulsion was bilateral, but more severe in the left side. After the fit there was conjugate deviation of the eyes to the right. In slighter attacks the convulsion was limited to the left side. At the onset he sometimes saw a flash of light. The onset of one severe attack was carefully noted as being first in the left side of the face with loss of consciousness, and this was followed by jerking of the head to the right and twitching of the mouth to the right, with deviation of the eyes to the extreme left. Then the right arm was drawn up and convulsed, and afterwards the leg, subsequently the limbs on the left side. Finally the head turned to the right, and the eyes to the extreme right.

In the beginning of 1879 sight had almost completely failed. Movement of the eyes to the left was attended by nystagmus. Hearing was impaired on the left side, the watch not being heard at all, while it was clearly heard on the right. The left side of the face was very weak, the forehead being smooth. Sensation on the face was a little impaired. The tongue deviated to the right. The left arm was quite powerless; the leg could still be moved, although feebly, but it was not rigid. The right leg could be moved freely. Sensation was not much impaired in the left limbs. The occipital headache was slighter, but it afterwards again became severe.

During the next few months he had an occasional slight fit, and sometimes a feeling of the room going to the left. In the spring he had a more severe fit, with unconsciousness, and convulsion only on the right side; the eyes and head deviated extremely to the right. Other right-sided fits occurred during the next two months; slighter attacks were limited to the face, and one of these was followed by a copious flow of saliva. Then continuous delirium set in. His wife was dead, but he asserted that he could see her practising prostitution with other patients in the ward. When assured that it was not so, although he was blind, his reply was, "Do you think I cannot believe my own eyes?"

In August there was alternate convulsion in the right and left side of the face, and then a severe convulsion on the right side. In September the delirium ceased. Smell was noted to be still deficient, but "better on the left side than on the right." Occasionally, however, he had an unpleasant subjective smell "like a strong liniment, almost taking his breath away." On the right side hearing was still good, and on the left the watch could be now just detected in contact; there was still often subjective ringing. Taste was slow; occasionally a subjective bitter taste was experienced. Sight was practically gone; occasionally he still had subjective sparks to the left. Movements of the eyes were feeble, and on looking to the left the right eye did not move beyond the mid position. Swallowing was slow; he choked easily and coughed much. The chest moved well on the right side, very little on the left. He spoke at first in an audible tone, but his voice soon died down to a whisper. The left arm was powerless, with no rigidity, but he complained of a dragging pain in it, and if moved away from his side he could not tell where it was. The right arm was moved freely, but was too feeble to act on a dynamometer. The left leg was powerless, but without rigidity, and passive movement caused much pain in it. The right could be well moved. The right knee-jerk was present, the left very slight, and the attempt to obtain it caused pain. There was no foot clonus. Sensation to pain and touch was defective in the left limbs, normal in the right. He continued in this condition, having a few fits, generally slight. In one the convulsion was in the right arm and slightly in both legs. On December 7 he died suddenly.

Post-mortem.—A tumour was found in the right hemisphere, but no morbid process in the left or in the pons. The growth lay between the optic thalamus and corpus striatum on the inner side and the convolutions of the temporal lobe on the outer. It was almost confined to the white substance, ceasing about $\frac{1}{12}$ in. from the grey substance of the temporal convolutions and of the island of Reil, but it had invaded the outer segment of the lenticular nucleus behind the middle of the corpus striatum, and had compressed the other segments. It had also invaded the posterior extremity of the optic thalamus for $\frac{1}{8}$ in. only and the thalamic substance was softened in an area of $\frac{1}{4}$ in., just behind the anterior extremity. A tongue of growth (fig. 1) appeared at the base between the crus and the uncinate gyrus, which was displaced outwards, but not invaded superficially. This projecting area of growth was $1\frac{1}{2}$ in. from before back, and $\frac{5}{8}$ in. wide. It had compressed the optic tract almost up to the commissure. Behind, in front of the corpora quadrigemina, the growth had invaded the crus, in an area the size of a split-pea, and thence had passed into the anterior and slightly into the posterior right quadrigeminal bodies, but had not crossed the middle line. The anterior limit of the growth was the neighbourhood of the grey matter of the convolutions of the island of Reil, opposite the middle of the corpus striatum. The highest

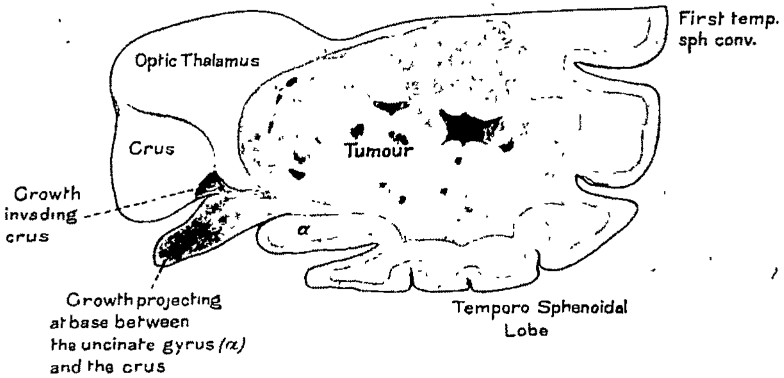

FIG. 1.
Section through the R. hemisphere, lower half, showing extent of tumour.

level it reached was a little above the floor of the lateral ventricle, outside it. It had filled the descending cornu, and extended beneath the anterior half of the posterior cornu, where it ceased by a rounded end, which was separated by a thin layer of softening from the cerebral substance. Nowhere else was there this separation; the growth was for the most part similar to the appearance and consistence of the grey matter of the cortex. An exception was presented by the region between the posterior part of the optic thalamus and the temporal cortex; here it was much firmer, and almost cartilaginous in consistence, yellowish in tint, and contained several small irregular cavities. It was evidently the oldest part of the growth.

The structure of the tumour had the aspect of a glio-sarcoma. It was composed of small oval cells, with a few fibres.

The features of this case are of great interest. The earlier fits must have been due to discharges in the right hemisphere, beginning with the auditory centre, as the result of the growth in the white fibres beneath it. Although the cortical centre would be isolated, the discharge would affect consciousness as a sound, and, extending to the motor region, which was not isolated, would cause convulsion on the left side. But before the convulsion the discharge seems to have spread to the centre for common sensibility in the ear, the external auditory meatus and concha, and thence round the neck and down the trunk to the left limbs. The discharge in the centre for common sensation in a special sense organ, as well as in that for the special sense itself, is rare, but is occasionally met with in epilepsy. I have not heard of it as a consequence of organic disease. It is especially intelligible in relation to the auditory centre, because the vibrations of touch stand in so close a relation to those of sound.

Lastly, a feature which is difficult to explain is the final convulsions on the right side, for which no explanation could be found in any lesion in the left hemisphere. We must consider that they were produced by the tumour in the right hemisphere acting on the left cortex through the fibres of the corpus callosum.

Colman [2], in a very instructive paper on hallucinations produced by organic disease, has published an account of a case of a tumour of the lower part of the central convolutions and the adjacent first temporal on the left side, in which there was the sound of bells in the right ear and afterwards a pleasant hallucination that a musical box was constantly playing. Colman has also referred to the case of a Jewess of Odessa who was subjected by her husband to blows on the head, which amounted to a rough physiological experiment, the results of which were described by Thomaschereski and Stamonowitsch in 1899. Congestion of the cranial bone, thickening of the dura mater and pia mater and inflammatory tissue beneath it, with changes in the cortex, were found over part of the right central convolution, the posterior half of the first temporal, the supramarginal and the angular gyrus. She suffered for long from convulsions, chiefly left-sided, and persistent auditory sensations on the left side, as well as left-sided visual hallucinations.

Last year an Italian widow, aged 39, was under my care at the hospital with symptoms of a tumour of the temporal lobe—double optic neuritis, headache, and convulsions, some preceded by the sound of a bell ringing on the left side, a very offensive smell, and the vision of a strange woman. Her case was especially fixed in our memory by the fact that she was quite willing that her head should be opened, but she had a profound objection, at first insuperable, to the removal of her hair; she only consented on the assurance that it need only be taken off on one side, and that it would surely grow again.. Sir Victor Horsley operated in July. A round encapsuled tumour, subcortical, was found in the temporal lobe; it reached down almost to the uncus. It was shelled out, through an incision, with as little injury to the brain as possible. She had afterwards slight left hemiplegia and diminution of sensation, but she left after two months, rapidly becoming well. Her only subsequent fits were on the day of the operation, and in these the previous aura, the offensive smell and vision of a strange woman, occurred after the cessation of ten minutes' spasm of the left arm, not before it.

These warnings had occurred only for a year and a half, when the fits had been more frequent, but the first fit was at 14, a month after her marriage, and they had occurred two or three times a year ever since, with tongue-biting and micturition, quite similar to those of the last year. No family tendency could be heard of. At the age of 19 she had a small tumour removed from the breast. The cerebral tumour was thought to be a glio-sarcoma; its structure suggested a long duration, and it may well have been present at the time of the first fit, and have caused the convulsions which were only associated with the auditory and olfactory warnings after it had attained a considerable size.

The reverse is the case with a woman who has just undergone the first stage of operation. Her age is 35. Fits have occurred for seven years and were associated long ago with a smell of rotten fish and also the sound of a voice, but these have been absent now for several years.

Their cause may be learned next week. [The operation proved it to be a large cyst, apparently connected with a lateral ventricle. The patient made a quick recovery.]

VISUAL.

Discharges in the visual centre seldom result from organic disease. The half-vision centre in the occipital lobe is still the only one generally recognized. Many years ago I suggested that the symptoms of disease give weight to the experimental conclusions of Ferrier, which indicated that the region of the angular convolution, between the parietal and occipital lobes, has a visual function. The facts suggest, I think, that this region is connected with both half-vision centres in a complex way, so as to represent the visual function of both eyes, but of the opposite eye to a greater extent than of the eye of the same side. Partial disease of this centre seems to lower its function as a whole. In the hemianæsthesia of hysteria this centre seems to be put out of action, and the effect on vision is well known. Such an hysterical condition is commonly regarded as not worthy of consideration, but functional states are the only evidence we can obtain of some arrangements in the central nervous system. A similar condition of vision may result from an organic lesion.

Some of the preliminary spectra of migraine can be best understood on the assumption that they depend on a slow discharge in this higher visual centre, on the side in which the subsequent headache is felt, especially those that have the form of what is termed the "angled fortification spectrum." The inhibition which accompanies the spectra seems sometimes to be seated in this centre, sometimes in both (when there is a central loss), sometimes in the related half-vision centre. But it is remarkable how very seldom inhibition of the latter forms part of the warning of idiopathic epilepsy.

I must not allow myself to be led into a discussion of functional affections, but I have to describe some cases of organic disease involving the angular gyrus and causing visual symptoms, cases which seem to afford definite support to the opinion I have put forward. I must again ask your pardon for the demand I shall have to make on your patience. The first case is important as evidence that the centre may also be concerned with word-blindness. Although it was diseased on the right side of the brain, the patient was left-handed except for writing, and in such a case the speech functions may have at least partial relations to the right hemisphere.

The patient was a man, James S., aged 45, who was admitted into the National Hospital, May 18, 1904. He was left-handed, and thinks he became so, except for writing, in consequence of an accident to the right hand at about 10. He had served in the Navy for twenty years, and during that time he had many blows on the head. He denied syphilis, but admitted many risks. Some time in 1902 he was struck on the head by a heavy piece of coal, and in November of that year—how long after the blow he does not know—he had a severe fit, which compelled him to stay in bed five days. He remembered, during this time, seeing a bright white light on his left. Subsequently he had slight attacks, in which his head turned to the left and there was brief loss of consciousness. In March, 1904, he had three severe fits in one day, with convulsion on the left side, and coloured lights to the left. Afterwards slighter fits occurred every few days until his admission, and afterwards.

He was a man of considerable intelligence. His smell, taste, sight and hearing were normal, and so were the optic discs and ocular movements. The left arm was slightly weak; the grasp being 75, compared with 90 on the right side, although he was in general left-handed. The left knee-jerk was the more active, and the plantar reflex was extensor, the right being flexor.

The attacks occurred frequently, but varied much in degree. Very careful notes were taken by Dr. Gordon Holmes, who was then resident medical officer. In the slightest the eyes became fixed without deviation. The left side of the face was the seat of slight tonic spasm, and the head turned slowly to the left; consciousness seemed dull for about a minute. In others, in addition, the left arm became rigid, and the elbow a little flexed without movement of the wrist or fingers. Smacking movements of the lips terminated each attack, but no sensation of smell could be ascertained, although sometimes he spoke of an unpleasant bitter taste in the mouth.

Many attacks, especially when more severe, were heralded by the appearance of several small balls, just to the left of the fixing point, and above, or below it. They were brightly coloured, red on the outside; within this was a zone of blue, the centre being white. They were always the same, and they moved to the left in a series of jerky movements, five or seven minutes after being first seen. During this time his eyes were constantly being jerked towards the left. Almost immediately after the appearance of the balls slight dimness developed in the left half of the field, which rapidly increased in degree, until it amounted to

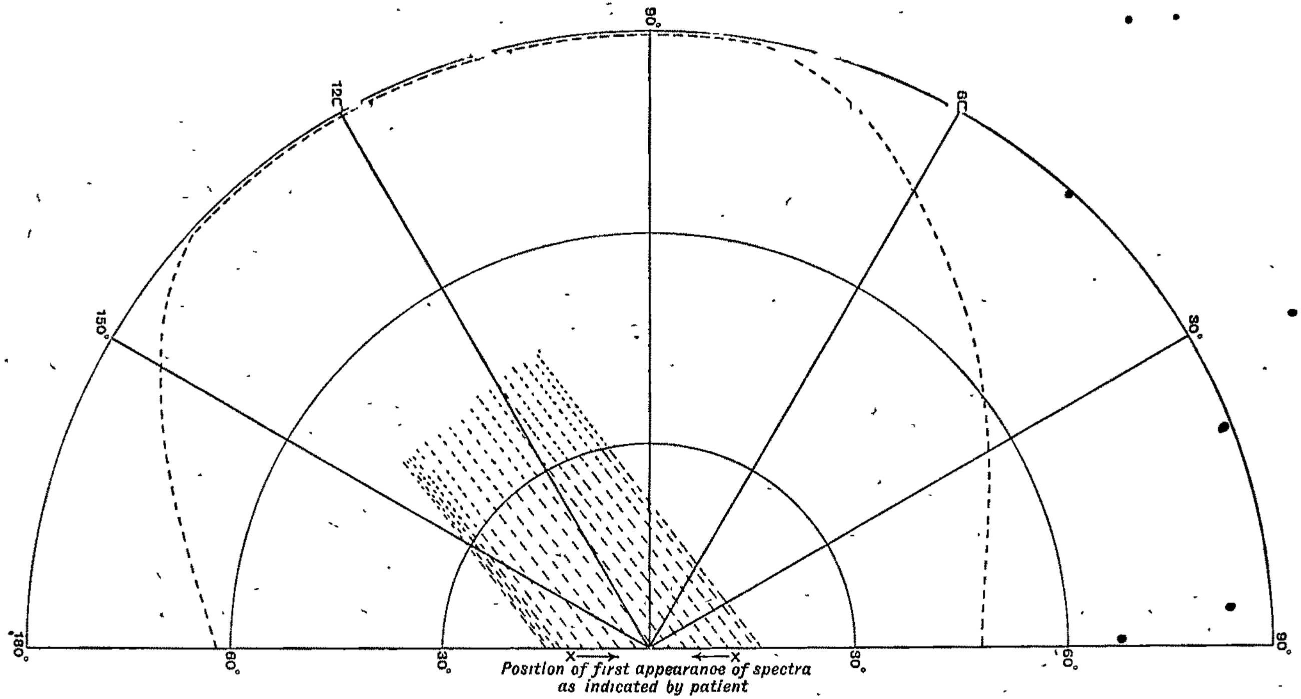
90°
120
60
150°
30°
180°
60°
30°
30°
60°
90°
X
X
Position of first appearance of spectra as indicated by patient

FIG. 2.

Explanation of Figures. (By Dr. Gordon Holmes.)

The four figures reproduced, as well as many others, were obtained by seating the patient in front of a wall on which a large sheet of white paper was fixed. He gazed at a black spot on the paper and indicated with a pencil the position and the direction of the movement of the spectral objects. An assistant who held a watch called out the time in seconds from the commencement of each attack when the patient indicated the position of his spectra; these times are marked on the last three figures opposite the points that the patient indicated. During the whole time he described the nature of the subjective phenomena and was frequently questioned about them.

The heavy lines represent the course of the coloured discs in figs. 3, 4, and 5, but where these lost their colour and appeared only greyish to the patient their course is shown by broken lines. In fig. 2 the first objects appeared at the points indicated by the crosses and moved downwards and outwards; later a larger number of similar objects appeared from the intermediate area nearer the fixation point and moved in the same direction; they lost their colour at about 30° from the fixation point.

A few of the patient's remarks on the number of the objects which he saw are enclosed in brackets on the diagram.

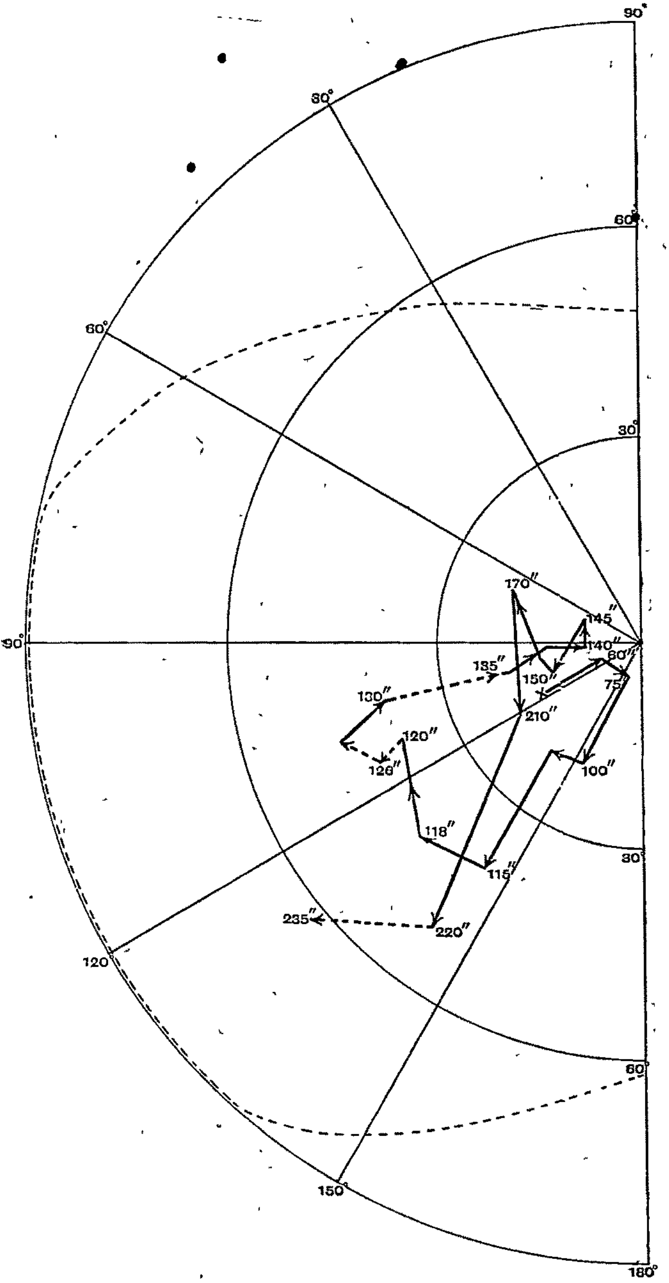
90°
80°
60°
30°
60°
90°
170″
145″
140″
135″
80″
150″
75″
130″
210″
120″
126″
100″
118″
115″
30°
235″
220″
120°
60°
150°
180°

Fig 3

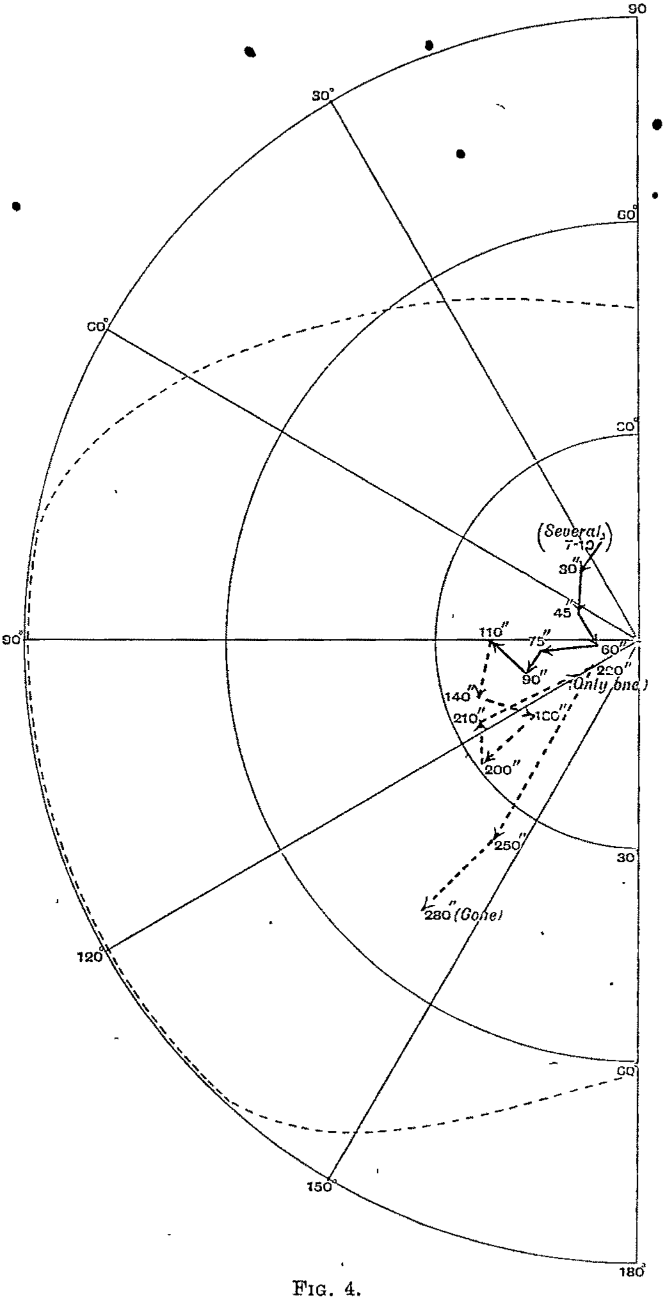
90
30°
60°
60°
30°
90°
(Several, 7-10)
30″
45
110″
75″
60″
90″
220″
(Only one)
140″
210″
160″
200″
250″
280″(Gone)
30
120°
60
150°
180°

FIG. 4.

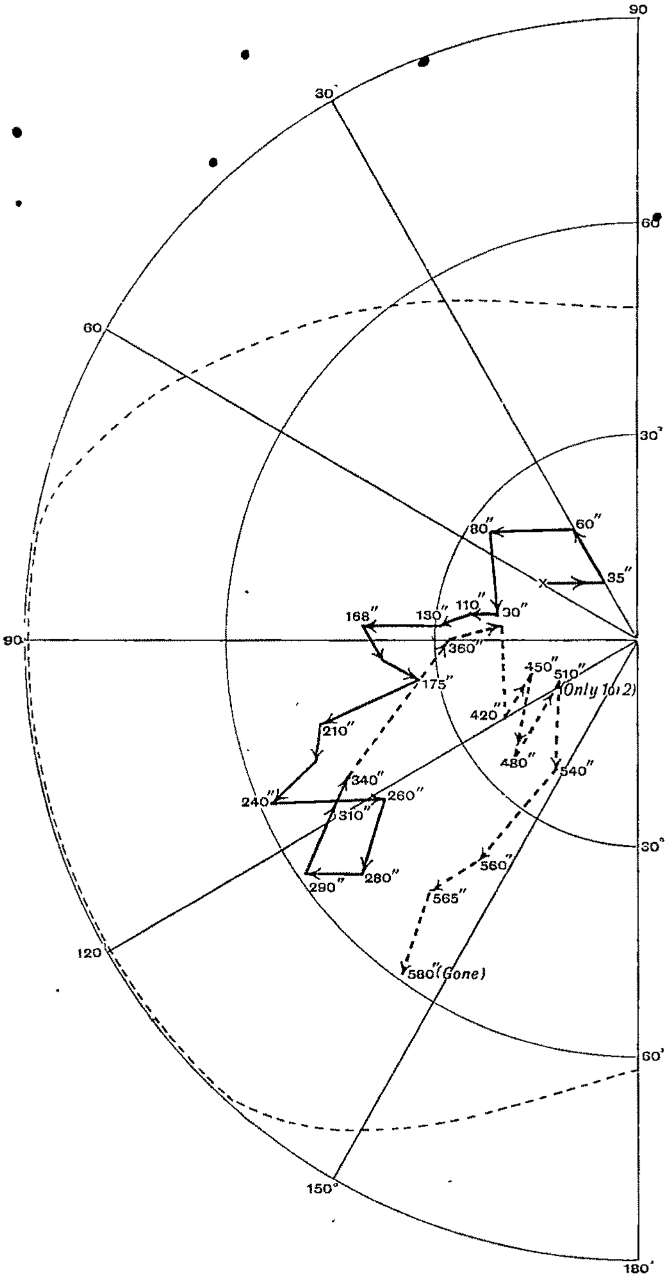
90
30
60
60
30
30
60
90
120
150°
180
80"
60"
35"
X
110"
30"
130"
168"
360"
175"
450"
510"
(Only 1 or 2)
420"
210"
480"
540"
340"
260"
240"
310"
560"
290"
280"
565"
580"(Gone)

FIG. 5.

complete hemianopia, extending to the middle line, except in a small area round the fixing point. It remained complete until about five minutes after the coloured balls disappeared, when it slowly lessened. Soon after the disappearance of the balls, round markings the size of the balls could be dimly seen at the place these had occupied. (The figures 2—5 represent the course of the spectra in some of the fits that were carefully observed.) The passing away of the hemianopia seemed to be from the medial part of the field towards the periphery, but a little dimness still remained half an hour afterwards.

In some attacks there was initial jerking of the eyes to the extreme left, and of the head, by tonic spasm. After a few seconds the spasm ceased, but presently came on again, and these recurrences went on for twenty minutes, each being attended by the noise in the mouth as if the patient were chewing; the arm and leg were rigid, and the arm was the seat of a fine tremor. In it there was complete loss of sensation, to touch and pain, up to two inches above the elbow-joint. The right limbs were normal. During this time the patient saw the coloured balls to the left, for about ten minutes. When they disappeared the eyes and head could be moved to the right. The tremor of the arm ceased, but was replaced for a time by irregular motion, comparable to athetosis. In a few minutes the loss of sensation had diminished, and extended only over the hand up to 2 in. above the wrist. Consciousness was unimpaired, but after an attack he frequently used wrong words in speaking, which bore no resemblance to those he intended to say.

Such an attack was sometimes followed by a severe recurrence. For instance, a quarter of an hour after one was at an end, he complained again of losing the feeling in his arm, which was found insensitive as before. The coloured balls reappeared, but soon changed to long coloured streaks in their previous course. Then he suddenly sank in a chair, saying that he had completely lost his sight, but in a minute he was again able to see as before. The attack passed off at the end of ten minutes. A third presently occurred of similar character, except that he did not again entirely lose his sight. A quarter of an hour later the fields were taken by a perimeter, and the hemianopia still persisted. Half an hour after the end of the last fit, the left arm was still rigid, and the leg abducted. By this time sensation in the arm had become normal.

Occasionally he felt the loss of sensation, which was verified by examination, and there was extreme deviation of the eyes to the left, without the appearance of the coloured balls. The fields were many times separately taken with the perimeter, and always found normal.

The following note was made of his impairment in speech: He always understands the nature of objects, and can easily express all his ideas and wants, speaking quite correctly. He understands everything said to him, even when spoken to quickly or in complicated sentences. He can read fairly well, but says it is much more difficult than it was before he became liable to fits; the effort tires him. He can see the words quite well, but has to read a sentence over and over again before he can understand the meaning. His right hand being good, he can write quite well and legibly, but complains of difficulty in expressing his thoughts in writing. He says: "I either leave out letters, or put in wrong letters, and sometimes I put in wrong words." When asked to write he began deliberately, stopping frequently to read over what he had just written, and often pausing to think how to write certain words. That which he wrote presented the mistakes in letters and words just mentioned. He can read fairly well what he has written, and recognises his mistakes, but cannot correct them. In writing from dictation he makes the same mistakes, but he can copy very well, or copy print into writing.

On July 5 he was trephined by Sir Victor Horsley. The opening in the skull was 3½ in. by 3½ in., and its centre was over the supramarginal gyrus. The dura mater there was very hypervascular, but not tense. On the 9th the second stage was carried out. The dura mater over the supramarginal gyrus was found, on incision, to be considerably thickened. It was removed up to near the edge of the opening. No tumour could be seen on the surface, but the pia mater was milky, opaque and thickened, and the surface of the brain was firm and nodular, and hard to the touch. A horizontal incision was made just above the supramarginal convolution, the substance of the brain was evidently sclerosed, greyish white and opaque in aspect; but, though the incision was deep enough to open the lateral ventricle, no tumour was found. In the angular gyrus, posterior to the incision, a firmer patch of sclerosis was found. The operation was well borne.

One or two fits occurred each subsequent day, on the left side, beginning in the face with clonic spasm. Consciousness was not lost. After a week they became less frequent. His mental state was very obtuse. During August there was steady improvement. The wound healed firmly, and his fits became less frequent and ceased by the middle of the month. In the middle of September it was noted that his mental state was good and bright, but now and then he seemed to become dull and strange, as though he had a slight attack of petit mal. Hearing was good, and the only defect in sight was a slight peripheral restriction in the left lower edge of the field. The two knee-jerks were equal, and the plantars both flexor. Spontaneous speech was quite good. He still complained of difficulty in reading, and that sometimes he could not understand what he read, but he read short sentences quite well and showed that he understood them. In reading aloud he made many mistakes, especially with the longer words, and even short ones were often pronounced wrongly. Spontaneous writing was with many mistakes, which he recognized on reading it afterwards. Writing from dictation was very imperfect, and long words he said he could not write, and, when urged to try, he was unable to produce anything like the correct word He seemed, indeed, to produce some retained visual symbols rather than to translate the auditory symbol into one of appropriate visual character.

By the middle of October all trace of hemiplegia had disappeared. He could write intelligently to his wife, but still failed in writing from dictation. He was discharged October 18, 1904, and has since been heard of as able to pursue his work in Ireland.

The visual phenomena present in the attacks of this patient seem best explained by a discharge of the assumed higher visual centre in the region of the right angular gyrus, which spread to the adjacent motor centres, or to it from them, and also produced temporary inhibition of the right half-vision centre, which very slowly passed away after the cessation of the discharge. They show how readily the half-vision centre undergoes inhibition, and that sometimes a secondary discharge of the centre in the other hemisphere may result, causing complete loss of sight. This has occurred when a hemorrhage in one occipital lobe has caused permanent hemianopia, but at first complete loss of sight by the inhibition of the half-vision centre in the other hemisphere, which, in one case I have seen, continued for a week.

Another case has been in the hospital, in which a traumatic superficial lesion of the angular gyrus gave rise to fits, beginning with a visual aura. The patient was under the care of Dr. Batten, to whom I am greatly obliged for the opportunity of describing it to you.

She is a girl, now aged 9, without neurotic heredity, who, when aged 3½ years, fell down fourteen or fifteen stairs and struck her head on the stone tiles of the floor below. There was no cut or bruise of the head, and the exact place of the blow was at the time uncertain. She seemed to have no loss of consciousness.

Half an hour later she had a fit, with loss of consciousness and twitching of the right side, face, arm, and leg, and she vomited several times during the convulsion. When it ceased she went to sleep, and after two hours woke up seemingly well, and no loss of power was noticed. Similar attacks recurred, and during the following year she had about forty. Then they ceased and she was free for three years, until Christmas, 1907, when they returned, and have frequently recurred since in severe form, until she came to the National Hospital on June 15, 1909.

In the hospital she had several attacks, differing in severity, but similar to those which had occurred previously. Each begins with a bright light in the right of the visual field, and she always rubs the eye with her right hand—not, she says, on account of any discomfort in the eye, but to get rid of the light; the head is then turned slowly to the right, and the eyes also deviate to the right. Consciousness is lost. Tonic spasm occurs in the right side, followed by clonic movement and twitching of the right side of the face and jerking of the eyes. The duration of the whole is two minutes. Consciousness then returns. Immediately afterwards there is loss in the right part of the field of vision, quickly passing away, and diminution of tactile sensibility in the right hand, and also extensor plantar reflex.

Some attacks were very slight and consisted only of the visual sensation, deviation of head and eyes to the right, and loss of consciousness, but with no movement of the limbs or clonic spasm of the face. The duration of such an attack was only about ten seconds, and after it no hemianopic loss could be found. Others were even more severe than that described above. One, which was also carefully observed by Dr. Gordon Holmes, began by the right visual spectrum and rubbing of the eye, with slow turning of the head to the right. On opening the eyes they were strongly deviated to the right. She indicated the position of the "twinkles," as she called the spectrum. as far out in the right visual field. Then she obviously became unconscious. For thirty seconds there was only strong tonic contraction of the muscles of the right side, the right arm being extended and the shoulder drawn downwards and backwards. Then clonic flexor spasm of the elbow occurred alone for about five seconds, when twitching of the right side of the face was added, strong jerking of the eyes to the right, and, almost simultaneously, clonic flexor spasm of the right fingers; and from this time the convulsion involved the face, arm, and leg, the trunk being also affected. No movement on the left side could be perceived. After two minutes the movements ceased, the last being slight twitching of the right corner of the mouth.

Immediately after the fit, sight was distinctly lost in the right side of the fields; it was soon regained, although for a time she said she saw objects more distinctly in the left than in the right half. Sensation in the hand was lessened to both touch and pain, and even after a quarter of an hour she complained that she could not feel the hand properly. The right plantar reflex was extensor after the fit, the left flexor, and the abdominal reflex was much less on the right side than on the left.

In one severe fit there was also some spasm in the left leg.

The visual spectra which she calls "the twinkles" always appear on the right, and she always rubs the right eye. They seem accompanied by deviation of the eyes to the right. She says the twinkles are "like stars." She occasionally says "they are all colours, green and red and blue and yellow." She says they do not move independently of her head.

Only on one occasion there seemed to be a difficulty in speech. About an hour after a fit her words are said to have been "all of a jumble" for a few minutes. At the time she could understand quite well what was spoken to her.

Apart from the attacks, her physical condition seemed quite normal. Her ocular movements were perfect, and so were all reflex actions, including the abdominal and plantar.

On July 6 she was trephined by Mr. Sargeant over the left parieto-occipital region, and, by saw-cuts, an osteoplastic flap was made and turned down. On exposing the brain the angular gyrus appeared paler and wider than the other convolutions, and, in its posterior part, was softened. On incision a dark grumous material escaped as well as some pale gelatinous substance. It seemed to be an old blood-cyst. It occupied a small region around the termination of the parallel fissure. The wound healed well. The attacks, however, returned; she has been readmitted, and they have ceased on treatment. They began with the same visual aura, but the convulsion had become bilateral.

I am indebted to Sir Victor Horsley for the facts of another case. An officer received a severe blow on the right side of the head in the hinder parietal region, which caused fits with a visual aura to the left. He was trephined, and laceration of the surface at the region of the angular gyrus was found.

In this connexion I may refer to the case I have already mentioned of the Odessa Jewess who had a persistent visual hallucination to the left, and the traumatic meningitis on the right side of the brain had involved the whole region of the angular gyrus.

These cases all speak with the same clear significance. It is different from the diminution of the remaining half-field in some cases of hemianopia, which was the evidence on which I chiefly dwelt in my Manual, in justification of the assumption in man of the higher visual function of the region of the angular gyrus. The higher is the level of function, the more complex its representation may be. We may hesitate to reject entirely that of which the evidence may still seem to be imperfect. The production of the same form of nerve energy by the various forms of dynamic force cannot but excite our marvel, and so must the difference in the effect produced in their perception. Especially is this the case in vision. The processes in the central structures must be very complex, and may well occupy an area more extensive than we can readily perceive.

BIBLIOGRAPHY.

[1] BUZZARD. *Lancet*, June 30, 1906, p. 1807.
[2] COLMAN. *Brit. Med. Journal*, May 12, 1894, p. 1016.
[3] JACKSON, HUGHLINGS. *Brain*, 1899, vol xxii., p 534.
[4] LINDE. *Monatschr. f. Pyschiat. u. Neurol.*, vii., No. 1, S. 44.
[5] LOCKEMANN. *Zeitsch. f. ration. Med.*, 1861, Bd. xii., S. 340.
[6] MILLS. *Journ. Amer. Med. Assoc.*, September 12, 1908.
[7] SOUTHARD. *Amer. Journ. Insanity*, April, 1908.
[8] SPENCER, W. G. *Proc. Roy. Soc.*, 1904, p 61.
[9] THOMASCHERESKI and STAMONOWITSCH. *Westnik Psychiatrii i Nevropatologie*, 1888, vi. (Russ.), and *Neurolog. Centralbl.*, 1889, S. 22.
[10] WESTPHAL. *Allg. Zeitschr. f. Psychiat*, 1863, Bd. xx., S. 485.

THE FUNCTIONS OF THE KINÆSTHETIC AREA OF THE BRAIN.

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SINCE the date of the discussion in 1886 before the Neurological Society of London[1] much new knowledge of various kinds bearing upon the question of the functions of the Rolandic region of the brain has been revealed. This problem is, therefore, now much more capable of receiving a definite answer, as evidence in regard to some essential points is no longer, as it was at that time, of a very fragmentary nature.

In effect, if not in actual words, one of the principal questions then considered was whether the so-called "motor centres" of the cortex were to be regarded as homologues of anterior spinal or of posterior spinal centres. For the support of the latter position it was needful to show that the regions in question were, in fact, regions for the registration of afferent impressions; and from another point of view that they were centres for the registration of such afferent impressions as are caused by movements of the body.

Evidence of this kind was at the time very scanty, but has now been fully brought forward by many workers, and notably by Horsley in his recent very important "Linacre Lecture" [15].

Three distinct views have been advanced in regard to the functions of this region of the brain. There is (1) the well-known view of Ferrier and many others, that the convolutions of this region contain actual motor centres for the production of voluntary movements; (2) the view adopted by myself and by Munk [25], to the effect that the so-called "motor centres" are in reality sensory centres for the registration of a complex set of impressions (kinæsthetic) occasioned by movements; while, lastly, (3) there is the view of Hughlings Jackson, adopted by Horsley, that the function of these convolutions is neither simply motor nor simply sensory, but is in reality "sensori-motor."

These are three definite views; but there is also a very indefinite, and to me meaningless, view suggested by some who speak of this region as containing "psycho-motor" centres. This metaphysical suggestion is neither helpful nor in any sense explanatory. It will be better, therefore, to cease to use a nomenclature which is not in harmony either with modern pyschological or physiological science.

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[1] *Brain*, April, 1887, on "The 'Muscular Sense', its Nature and Cortical Localization."

In regard to the first view, my adverse opinion has been several times expressed between 1880 [3] and 1902 [4]—at periods, that is, when both the pre-central and the post-central convolutions were supposed to contain motor centres.

The object of the present communication is rather to show that the second and third views are quite different from one another, and that they have been arrived at from a wholly different standpoint, depending mainly upon opposite views concerning (*a*) the nature of "muscular sense" impressions, and (*b*) their seat of registration in the brain.

It seems needful that a statement to this effect should be made owing to the fact that Horsley in his recent lecture, while favouring the name of "kinæsthetic area" applied by me to this Rolandic region, strangely enough seems to think that my view is identical with that of Hughlings Jackson, as the following quotation will show ([15], p. 125):—

> "It is time that yet another protest should be raised against the expression 'motor area' as untrue scientifically, and, like many other unfortunately convenient expressions, so misleading as to hamper the progress of knowledge.
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> "Dr. Bastian's much more logical expression 'kinæsthetic area' has, unfortunately, not found general adoption, and yet none better expresses the fact that there is no such thing as a purely motor centre in the cortex cerebri, the whole structure being, in Dr. Hughlings Jackson's language, sensori-motor, or a combined mechanism for the record and execution of afferent and efferent nerve impulses."[1]

This quotation, with what follows, gives some indication why Horsley adopts the "sensori-motor" view, seeing that, as a reason against the expression "excitable" area also often used, he speaks of the visual area on stimulation giving "motor, that is efferent, results on stimulation," though he apparently, in the main, relies upon an *a priori* dictum of Hughlings Jackson, as we find him saying ([15], p. 126):—

> "The true method of regarding the anatomical constitution of the cortex cerebri should begin by accepting the principle first enunciated by Hughlings Jackson from the consideration of the nervous system from the evolutionary

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[1] When I introduced the word kinæsthesis ("The Brain as an Organ of Mind," 1880, p. 543) it was as an equivalent for "sense of movement," from *κινέω* to move and *αἴσθησις* sensation, and it has never been used by me in the sense implied in the above quotation.

standpoint—namely, that every centre in the nervous system must be sensori-motor.

Neither of these reasons, however, appeals to me. The *a priori* theoretical dictum of Hughlings Jackson seems to me entirely unproven, and indeed at variance with well-known facts. If it is meant that a sensory centre never exists without being in connexion with a motor centre, and *vice versa*, I entirely agree; but to say that every centre must be sensori-motor tends, as I submit, to convey an erroneous impression, because the separate components of the functional couple may not be contiguous, or so as in effect to form one centre; the sensory and the motor parts of the couple may be, and often are (both in lower animals and in man), wholly separate, and even comparatively remote from one another, though connected by internuncial fibres.

Of course, the stimulus which awakens the activity of a motor ganglion or centre *must* come to it through connecting fibres from a sensory ganglion, centre, or knot of cells. That happens in every reflex action. But because the stimulus issues from a sensory centre, and is therefore "efferent," that gives no justification for speaking of such an efferent stimulus as "motor" any more than it justifies us in regarding the sensory centre from which it proceeds as a "motor" centre. Yet we now have Horsley speaking of stimulation of the visual area giving "motor, that is efferent, results," just as in 1876, in his "Functions of the Brain," Ferrier contended that cortical centres "immediately concerned in effecting volitional movements" are, "as such, truly motor." In each case the important point is lost sight of—that "efferent" currents issue from sensory as well as from motor centres, and that it has never been customary to speak of the currents issuing from a sensory centre as "motor" currents, even though it is true that their ultimate result is to evoke motion.

Still, it is clear that the mere fact that movements can be produced as a result of stimulation seems to influence Horsley in speaking of various parts as motor, when we find him saying ([15], p. 126), "We may be certain, therefore, that the more essentially motor part of the kinæsthetic representation of the upper limb lies in the gyrus in front of the fissure of Rolando," and when we find the sole reason given in support of this statement is that this region when stimulated is followed by specialized movements of the upper limb.[1]

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[1] In other places also he speaks of "the whole efferent or motor area," and of "an efferent or motor cell," as if the two terms were legitimately interchangeable.

It is true, as Bevan Lewis showed in 1878, that this pre-central gyrus contains giant pyramidal cells of Betz in groups, and that such cells have been commonly regarded, owing to their large size and to their resemblance to cells in the anterior cornua of the spinal cord, as having motor functions. This, however, is a most inconclusive and untrustworthy point of view. As Horsley says, and as was originally pointed out by Bevan Lewis, it is an anatomical rule that such a cell "varies in size according to the distance the axone has to travel in the central system—for example, the largest pyramids in the pre-central gyrus are in the leg area," while in centres concerned with movements of "the face, larynx, pharynx, and eye muscles, as well as part of the representation of head movements," no such large cells are to be found, but only medium and small pyramids. Such pyramidal cells of different sizes are regarded by Horsley as "outgoing stations in any part of the cortex, because their axones pass down through the central nervous system." That being so, we are in the face of valid evidence that they are "outgoing stations"—that is, so many points of origin for pyramidal fibres. Such a conclusion, however, in no way helps to decide the question as to the nature of the centre from which they proceed. On the other hand, he says, "the much more numerous small cells of polygonal and varying outlines, forming the granule layers, may be regarded as belonging to a mainly afferent" system; and this view of their nature, which Horsley put forward in 1888, has, he adds, "now received the adequate criticism of research, and the receptive character of the cells may be considered to be established by the reseaches of Bolton, Ramon y Cajal, Mott, Watson, and von Kappers, among many others."

Now, as I pointed out in 1880 [5], and reinforced in 1886 in the discussion before the Neurological Society, the experimental evidence cited in favour of the existence of motor centres in the cortex is absolutely inconclusive in face of my view that such centres are seats of registration for kinæsthetic impressions, when associated with the opinion also expressed as to the relation of such impressions to the initiation of volitional movements. It was submitted that purely sensory centres of this type ought to give rise to specialized movements on stimulation, while, on the other hand, excision of such centres should produce actual paralysis—that is, just such effects in each case as had led to their being regarded as motor centres.

The nature of my views as to the importance of kinæsthetic centres in the production of voluntary movements, and the precise part which they play, have been fully expressed elsewhere [6], and need now be only briefly referred to.

Many simple voluntary movements, as we know, occur with all the rapidity and ease that characterize so-called "ideo-motor" movements. At other times our voluntary actions only take place after more or less complicated preliminary processes of deliberation or weighing of motives. So that, as Hartley long ago said [13], "The Will is, therefore, that desire or aversion which is strongest for the present time"; or, as William James puts it [21], Volition is "the stable victory of an idea although it may be disagreeable, the permanent suppression of an idea, although it may be immediately and urgently pleasant"

Thus, instead of the simple passage of an impression from the afferent to the efferent side of a reflex spinal arc, we have in the cerebrum, in the case of voluntary movements, on the afferent side a widespread activity in many regions of the cerebral cortex, resulting in a more or less complex process of deliberation.[1] So that the act of willing a particular movement consists essentially in a consent (after balancing of reasons that may exist for or against) to the occurrence of such a movement, the movement itself being at the same time mentally prefigured by certain revived sensations—such revival representing, as James Mill [24] said long ago, "the last part of the mental operation," a view which has been adopted by William James [22] as well as by myself.[2]

This last link in the chain of association, which is to be immediately followed by the occurrence of the movement, is a revival in idea (or a conception) of the movement to be executed—that is to say, a memorial recall, more or less unconscious, in kinæsthetic and other centres, of the sensations previously experienced on the occurrence of such movements; the revival in the case of limb movements being in visual and in kinæsthetic, and, in the case of speech, in auditory and kinæsthetic centres—the last and most essential portion of the revival being in each case in the kinæsthetic centre.

But immediately following upon this last link in the chain of association, that is, of memorial revival, when consent is given (and no inhibition comes into play) there occurs the outflowing stimulus from this sensory centre probably into the pyramidal cells, large or small, and thence along the cortico-spinal or cortico-bulbar constituents of the pyramidal tract to the related real motor centres. We should have, in fact, taking place here a process more complex, but otherwise similar to that which occurs when a stimulus passes from the sensory side of a simple spinal arc to its associated set of motor cells. In the case of the execution of voluntary movements there is great complication on the sensory side, for as Spinoza has said, "Voluntas et intellectus unum et idem sunt"; and more or less activity of intellect must come into play before the outgoing stimulus is permitted to pass. There is, however, no room or occasion here for the action of motor centres—not, that is, till we come to the actual execution of the movement, all the cell and fibre mechanisms for which have been previously, or are being, slowly built up in the spinal cord and in the bulb. This latter view, concerning spinal co-ordination, originally taught by Volkmann and Müller about 1844, has of late been strongly supported by Exner [12], Sherrington [27], and also by Beevor [11]. The latter, for instance, came to the conclusion that "all the evidence is in favour of the linkage of the ultimate constituents of a movement being in the spinal cord."

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[1] As to this process the philosopher of Malmesbury said. "The whole sum of desires, aversions, hopes and fears, continued till the thing be either done or thought impossible, is what we call Deliberation."

[2] Thus Wm. James says: "Every representation of a motion awakens the actual motion which is its object, unless inhibited by some antagonistic representation simultaneously present to the mind." Munsterberg has also expressed similar views.

A certain amount of cerebral co-ordination, dependent upon the activity of cortical sensory centres must, of course, be called into play in learning new movements. And by brain activity we habitually regulate the force, the rapidity, or the combinations of movements which are organically represented in the spinal cord, so as to make our actions precisely accord in quality with the aims conceived.

Now, as already intimated, when the discussion took place in 1886, and subsequently, it was denied by many that valid evidence was forthcoming for the existence of sensory functions of a kinæsthetic, or, indeed, of any other type pertaining to the Rolandic convolutions; and yet for the establishment of my view it was, of course, most important to show that such evidence did exist. I showed that the experimental evidence could be explained just as well in accordance with my interpretation of the functions of the Rolandic area, as with the view that it contained motor centres. But that was not enough, the proof of the existence of sensorial functions was for me all-important. Now, however, through the prolonged investigations of Horsley and many others, including Mills, Ransom, Mott, Campbell, Brodman, Bonhoeffer, and Cushing, there is no further room for doubt on this subject. Horsley, while referring to the conclusions of the other workers above mentioned, says in regard to his own investigations ([15] p. 127) :—

"Every lesion in this region in man that I have seen during the past twenty-four years has served to confirm Bastian's and Munk's mode of regarding cerebral function."

This uniformity of result is probably in part attributable to the extreme care and judgment with which Horsley conducted his tests for the sensory defects in question. He has come to the conclusion, for instance, as he tells us, that "the sensory representation of the upper limb extends over a wide area of the cortex—namely, at least both Rolandic gyri."

Thus, the final evidence needed to support my view and that of Munk is now forthcoming—namely, that the kinæsthetic area is an aggregate of purely sensory centres, and that the cortico-bulbar and cortico-spinal fibres which issue from pyramidal cells in the pre-central gyrus (or elsewhere) are simply internuncial fibres for the conveyance of appropriate stimuli to true motor centres in the bulb and in the spinal cord.[1] Whenever impulses pass from sensory to motor centres they must pass through such internuncial fibres, and, of course, every sensory centre, whether it be in the brain or in the spinal cord, must be in relation not only with afferent but with efferent fibres.[2]

And as regards the centres themselves, situated in the kinæsthetic area, they are believed by me to be the seats of registration of complex sets of afferent and habitually associated impressions, such as must always stream up to the brain when this or that part is in motion—impressions from muscles, tendons, ligaments, articular surfaces, and skin, which together are spoken of as kinæsthetic impressions [7]. Such centres must be brought into intimate functional relation, not only with visual and auditory, but also with others, for the initiation, in ways already referred to, of all our innumerable muscular movements; while the nervous mechanisms concerned with the actual performance of these correlated muscular actions have been shown to be situated in the bulb and in the spinal cord.

I am quite prepared, therefore, to say with Victor Horsley that the pyramidal cells, large and small, probably constitute the "outgoing stations" of the kinæsthetic area, and that the cells of polygonal and varying outline may in the main be "receptive" elements. But if as he has shown, the Rolandic gyri are centres for afferent impressions, I must altogether demur to the propriety of speaking of the pyramidal cells as "efferent or motor cells," and of the particular part of the area in which they are situated as an "efferent or motor area."

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[1] I have always used the term "internuncial" for fibres connecting sensory with motor centres, reserving the term "commissure" for the connexions of centres of like kind, either sensory or motor.

[2] In his valuable work on "The Localization of Cerebral Function" (pp. 105 and 205), A. W. Campbell adduces two main reasons against the views here adopted: (1) The absence of evidence that lesion of the pre-central area is alone adequate to produce "paralysis of the muscular sense"; and (2) the existence of anatomical evidence that muscular sense-impressions are first conveyed to the post-central area of the cortex. Both these objections have now been nullified.

To argue that groups of cells have motor functions merely because stimuli issuing from them evoke movements when they impinge upon motor ganglia, is quite on a par with the argument that an organ has sensory functions because fibres come to it from sensory cells, as Foville and others originally contended in regard to the cerebellum.

Horsley uses the language before mentioned, and speaks of the whole kinæsthetic area as a "sensori-motor" region apparently in part because he would seem for the moment to lose sight of the fact that every sensory centre must have efferent fibres, but mainly because he appears absolutely to accept as true the *a priori* dictum of Hughlings Jackson that "every centre in the nervous system must be sensori-motor." This doctrine has also been accepted by others; but, as already intimated, I venture to think it can be shown to have a merely speculative basis, and to be, in fact, at variance with much existing knowledge. That a sensory centre never exists which is not in relation with a motor centre, and that a motor centre never exists without being in connexion with a sensory centre, is a truism; but that is quite different from the dictum to which Horsley gives his adhesion.

It is, of course, true that parts of the kinæsthetic area are "outgoing" stations, while other and much wider regions are "receptive" stations, partly perhaps lying above the former, and partly more fully occupying other contiguous regions. The outgoing, or so-called "motor," stations have been shown by Sherrington and Grünbaum (whose observations have been confirmed by Page May and Gordon Holmes, and by others working in different ways) to be mainly, though perhaps not wholly, limited to the pre-central gyrus. Other portions of the kinæsthetic area are now held, on clinical evidence, to include the bases of the three frontal convolutions (the so-called "eupraxic centres" of Liepmann), as well as the post-central gyrus, and possibly portions of the parietal convolutions.

When we consider the enormous importance of the kinæsthetic centres for the initiation of all the movements of which we are capable, it is easy to understand why the receptive and co-ordinating portions of these centres should be so extensive; while, on the other hand, it seems equally clear that there must be definite regions from which the emissive fibres are to issue. Whether, however, such fibres are to issue from many parts of the general centre, or whether only from some definite portions of it (as is mostly found to be the case), cannot be considered to be a point of any importance in its bearing upon the general question concerning the nature of the functions carried on in this region of the cortex.

It becomes needful now to endeavour to discover the reasons that have led Hughlings Jackson to adopt not only his before-mentioned view that "every centre in the nervous system must be sensori-motor," but that "mental operations in the last analysis must be merely the subjective side of sensory and motor substrata" [16]. This inquiry is especially needful because the doctrine that motor centres exist in the cerebral cortex is undoubtedly traceable to his teaching, and was started as a sequence of his acute and masterly clinical observations on certain forms of convulsion (now named after him), together with his conclusion that they were due to "discharging lesions" in portions of what he terms "the middle level of evolution." He says [17], "I supposed the particular parts diseased to lie within the region of the middle cerebral artery," so that, as Hitzig said in the second "Hughlings Jackson Lecture" [14]:—

> "By referring the convulsions to such centres he argued the existence of motor centres in the brain, and even told to a certain degree what their position was before physiological research was able with certainty to prove where they were."

Subsequent speculations on the evolution of the nervous system led Jackson to formulate some very definite hypotheses which may best be given in his own words. He says ([18] p. 65):—

> "The Rolandic region of the cortex cerebri and the prefrontal lobe are the motor provinces of, respectively, the middle and highest levels [of evolution]. . . . . In what follows it is to be understood that the unit of constitution of the whole nervous system (not excluding the highest level, the so-called 'mental centres') is sensori-motor; and also that in the middle and highest levels, at least, the so-called motor provinces are only *chiefly motor*, and the sensory provinces only *chiefly sensory*."

These views are, as I have said, of the nature of speculations or hypotheses, and, so far as I am aware, there is no positive evidence that can be adduced in favour of what he says concerning the existence of motor centres in the prefrontal region, and only, as we have seen, most questionable and inconclusive evidence for the existence of motor centres in the Rolandic region—his so-called "middle level of evolution."[1]

In regard to his view that every centre in the nervous system must be sensori-motor, facts of a clearly contradictory nature have already been referred to; while an altogether opposite interpretation of the clinical and experimental evidence upon which the supposed existence of motor centres in the Rolandic region had been based has been given and several times repeated since 1880, when this subject of the supposed existence of motor centres in the cerebral cortex was pretty fully discussed in two chapters of my book, "The Brain as an Organ of Mind"—namely, that on "Will and Voluntary Movements" and that on "Cerebral Mental Substrata."

Jackson, however, in the face of the objections alleged, has been content merely to reiterate his speculative views, without attempting to produce actual evidence in support of them or to refute adverse criticisms.[2]

There remains for consideration, however, the important question whether it is true, as he affirms, that "mental operations in the last analysis must be merely the subjective side of sensory and motor substrata." What is the basis for such a belief?

We may freely admit with him that the main elements of that part of mind known as intellect is carried on as "visual and tactile ideas and words" [18].

Then, again, it is undoubtedly true that the impressions coming from every one of the "special" sense organs are, in part, dependent for their various combinations upon the movements of such organs and of the parts in which they are situated. For this as well as for other reasons the connexions existing between the visual perceptive centre and the kinæsthetic centres must be peculiarly intimate for the initiation of all movements of limbs; just as, in the case of the complex movements concerned in articulate speech, their promotion is dependent upon the regulative activity of combined auditory and kinæsthetic centres.

Now, however, comes the all-important question in reference to these views of Hughlings Jackson. Is it or is it not true that muscular sense impressions are, as Bain put it, "concomitants of the outgoing current"? Are we to believe, with him, that "the sensibility accompanying muscular movement coincides with the outgoing stream of nervous energy, and does not, as in the case of pure sensation, result from any influence passing inwards by the incarrying or sensitive nerves"? [1].

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[1] In regard to his "highest level" in the prefrontal region he has said [19]: "I admit that this part of my scheme is very hypothetical; and I do not suppose that there is so decided an anatomico-physiological difference between the motor provinces of the highest and middle levels as there is between those of the middle and lowest levels."

[2] See his contribution to the discussion from the presidential chair in 1886 (*Brain*, April, 1887, p. 107).

This view has never been accepted by me, and I had some controversy with Professor Bain on the subject in *The Fortnightly Review* in 1869, which was followed by an article "On the Muscular Sense and on the Physiology of Thinking" [9], in which this question was gone into as fully as then existing knowledge permitted, with the result that much evidence was adduced against Bain's view.[1] On the other hand, Jackson has always sympathized with this doctrine; and when we look to the way in which it was developed by Bain, it becomes easy to see some at least of the considerations which must have influenced Jackson in arriving at his before-mentioned view concerning the ultimate nature of mental operations.

Bain, for instance, says that "a suppressed articulation is, in fact, the material of our recollection, the intellectual manifestation, the idea of speech"; while Hughlings Jackson has habitually spoken of the ideas of words as "motor processes." Then, again, we find Bain saying [2]:—

> "In all that regards visible movements and visible form, the muscular consciousness, it is now contended, is the indispensable element, the optical sensations merely guiding the movements. Naked outlines as the diagrams of Euclid and the alphabetical characters are, to say the least, three parts muscular and one part optical."

But when we recollect that, in Bain's view, muscular sensations are realized only in motor centres, we can easily understand why Hughlings Jackson, as a believer in his doctrines, should urge, as he has done, that "mental operations in the last analysis must be merely the subjective side of sensory and motor substrata."

On the other hand, in the light of opposite views concerning the nature of muscular sense-impressions, such a statement would receive no support whatever, seeing that, in accordance with this doctrine, all the sensations resulting from movement are afferent impressions, and as such are registered in sensory centres.

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[1] I have previously bracketed the name of Wundt with that of Bain as an upholder of very similar views; the propriety of this, however, was questioned by De Watteville in the 1886 discussion, and I have since found that his views are not so extreme as those of Bain. While postulating the existence of "feelings of innervation" both with what he terms internal and external volition, he now admits that the sensations of movement are due to impressions carried by afferent nerves ("Psychologie Physiologique," 1886, t. ii, p. 445).

It is useless now to attempt to revive this old controversy as to the nature of muscular sense-impressions. The views of Bain were put forward before the sensory fibres and apparatus in muscles had been discovered, and probably never would have been brought forward had he known of their existence. As F. E. Batten has pointed out, [10], it was not till 1888 that the real nature of the muscle-spindle became known, and not till 1895 that the final proof was given by Sherrington that the nerve fibres from these bodies pass up in the posterior roots. Still, even apart from that, very strong evidence condemnatory of the view could be and was adduced [9]. It is now, indeed, almost universally admitted that muscular sense-impressions, and those resulting from movements generally, instead of being "concomitants of the outgoing current," and as such realizable in motor centres, are all of them afferent impressions occasioned by, and emanating from, the parts in movement.

The different point of view that has now been brought about may be illustrated by a comparison of statements made by Ferrier in different editions of his "Functions of the Brain." Thus, in the first edition of 1876, he said :—

> "The motor centres of the hemispheres, besides being the centres of different movements, are also the organic basis of the memory of the corresponding movements, and the seat of their re-execution or ideal reproduction . . . . Ideal movements form no less an important element in our mental processes than ideally revived sensations (p. 266).

But in the second edition of this same work (1886) we find the author renouncing the above views (which were in harmony with those of Bain and Hughlings Jackson) as being no longer tenable. While still adhering to his view that in the cortex there are motor centres as well as sensory centres, he adds :—

> "But whereas we have sensory ideation in and by itself, we have no ideas of movement apart from the sensory centres through which the activity of the motor centres is revealed in consciousness" (p. 436).

Then, again, Münsterberg [26] as well as James [23] has most carefully considered the evidence that had been adduced in favour of "feeling of innervation" and muscular sense impressions as "concomitants of the outgoing current," and they have both come to the conclusion that there is no real evidence in support of either of these doctrines. James has, moreover, specially considered "The Ambiguous Import of Eye Movements ([22] pp. 234-37) in reference to these problems, and finds nothing there to shake the general consensus of evidence, although Hughlings Jackson and L. Paton have recently opened up a discussion of this question upon the basis of "Some Abnormalities of Ocular Movements" [20]. I cannot, however, consider that the evidence on which they rely in one special case is either unambiguous or capable of in any way weakening the general evidence against the doctrine which they favour.

The views to which I have arrived are therefore, as will have been seen, wholly different from those of my friend Dr. Hughlings Jackson. I do not regard all nerve centres as "sensori-motor," as Horsley implies; I do not believe that motor centres of any kind exist in the cerebral cortex; I do not believe that conscious ideal recall ever takes place in any motor centre; nor can I therefore agree that "mental operations in the last analysis must be merely the subjective side of sensory and motor substrata." I am driven to believe that the doctrine of Bain concerning the muscular sense is altogether unfounded, and that with it should vanish what alone could afford a positive basis for the doctrine of Hughlings Jackson, to which I have last referred.

From my point of view, as I said in 1880 [8]:—

> "The cerebral cortex is to be regarded as a continuous aggregation of interlaced 'centres' towards which ingoing impressions of all kinds converge from various parts of the body; here they come into relation with one another in various ways and conjointly give rise to nerve actions which have for their subjective correlatives all the sensations and perceptions, all the intellectual and all the emotional processes which the individual is capable of experiencing. From these terminal and complexly related 'end stations' for ingoing currents and from certain annexes in connexion therewith outgoing currents issue. . . . The plan on which nerve centres generally are constructed, of whatsoever grade, makes it essential that the stimulus which awakens the activity of a 'motor' ganglion or centre shall come to it through connecting fibres from a sensory ganglion, centre, or knot of cells—that is, from cells which stand in immediate relation with ingoing fibres."

I do not therefore think that the kinæsthetic centres in the cerebral cortex are any the less worthy of the name of sensory centres, simply because their emissive or outgoing fibres pursue a long course in a downward, rather than in a horizontal or upward, direction, and are found to be embodied in the "pyramidal tract." And, apart from their importance in regard to the initiation of voluntary movements, they have an importance of a different kind, since their impressions enter inextricably into a large majority of our mental processes, as widely and as inextricably, in fact, as the assumed "motor substrata" of Bain and Hughlings Jackson were deemed by them to be intertwined with what Bain distinguished as '"passive,' sensibilities."

I have further expressed the view that "motor substrata" really lie altogether outside the domain of consciousness ([5] p. 599.)

> "Motor centres, wherever they may be situated, are parts whose activity appears to be wholly free from subjective concomitants. No 'ideal' reproductions seem ever to take place in such centres; they are roused into activity by outgoing currents, and, so far as we have any evidence, the induction in them of molecular movements which, immediately afterwards, issue through cranial and spinal motor nerves to muscles are simply physical phenomena. These processes are as free from subjective accompaniments as are the actual molecular processes thereby induced in the muscle itself. It is the altered condition of the muscle thus induced, and of contiguous parts as occasioned by the movement, which together engender a body of ingoing impressions, the terminus for which is the kinæsthetic centre."

Whenever psychologists and physiologists generally may, like James, Münsterberg, Ferrier and Ross, come to hold opinions of this kind in regard to the absence of any immediate psychical accompaniment resulting from the activity of motor centres, it will soon be more widely recognized that there is not the slightest need for postulating the existence of "motor" or of "sensori-motor" centres in the cerebral cortex.

Bibliography.

[1] Bain. "The Senses and the Intellect," 3rd Edition, p. 76.
[2] *Idem.* *The Fortnightly Review*, April, 1869, p. 498.
[3] Bastian, Charlton. "The Brain as an Organ of Mind," 1880, pp. 543, 548-557, 591-600.
[4] *Idem.* Presidential Address: "On the Neural Processes Underlying Attention and Volition," *Brain*, April, 1902.
[5] *Idem.* "The Brain as an Organ of Mind," pp. 584-88.
[6] *Idem.* *Op. cit.*, pp. 548-588, and *Brain*, April, 1892, pp. 23-34.
[7] *Idem.* *Op. cit.*, pp. 540-544, and pp. 591-594.
[8] *Idem.* *Op. cit.*, p. 584.
[9] *Idem.* *Brit. Med. Journ.*, May, 1869.
[10] Batten, F. E. "The Muscle-Spindle under Pathological Conditions," *Brain*, 1897, vol. xx, p. 138.
[11] Beevor, C. E. "The Co-ordination of Single Muscular Movements in the Central Nervous System," *Journ. of the Amer. Med. Assoc.* vol. li, pp. 89-95.
[12] Exner, Sigmund. "Zur Kenntniss der Wechselwirkung der Erregungen im Centralnerven system," *Pflüger's Archiv*, 1882, xxviii, s. 487.
[13] Hartley. "Observations on Man," 1748.
[14] Hitzig. *Brain*, 1900, p. 547.
[15] Horsley, Victor. *Brit. Med. Journ.*, July 17, 1909.

[16] JACKSON, HUGHLINGS. "Clinical and Physiological Researches on the Nervous System" (Reprint), 1876, pp. 20-37.
[17] *Idem.* "Study of Convulsions," *Brain*, April, 1886.
[18] *Idem.* *Brit. Med. Journ.*, 1898, vol. i, p. 65.
[19] *Idem.* *Lancet*, Jan. 8, 1908, p. 80.
[20] JACKSON, HUGHLINGS, and PATON, L. "On Some Abnormalities of Ocular Movements, &c.," *Lancet*, March 27, 1909, p. 909.
[21] JAMES, WM. "The Feeling of Effort," 1880, p. 22.
[22] *Idem.* "The Principles of Psychology," 1890, vol. ii, pp. 486-495.
[23] *Idem.* *Op. cit.*, pp. 493-495, 499-505, 514.
[24] MILL, JAMES. "Analysis of the Human Mind," 1830, ii, p. 266.
[25] MUNK. "Ueber die Functionen der Grosshirnrinde," 1881.
[26] MUNSTERBERG. "Die Willenshandlung," 1888, s. 73, 82, 87-88, and *Mind*, 1888, p. 463.
[27] SHERRINGTON, C. S. "On Reciprocal Innervation of Antagonistic Muscles." Seventh Note. *Proc Roy. Soc.*, 1905, B, lxxvi, p. 162.

CEREBRAL MONOPLEGIA, WITH SPECIAL REFERENCE TO SENSATION AND TO SPASTIC PHENOMENA.

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CHAPTER I.—INTRODUCTION.

THE observation made by my teacher, Professor Petrén, some years ago that certain cerebral lesions—viz., those of cortical origin—may give rise to a paralysis without the tendon reflexes necessarily being increased induced me to undertake the following investigation. On his initiative I have attempted to investigate this condition on a larger material. It was assumed that this peculiarity of cortical lesions depended on sensory disturbances, more especially of the muscular sense. I have been unable to confirm this hypothesis, but the question led me to a closer study of the sensory disturbances that result from cortical lesions. The aim of this investigation is to study in detail on a large material the symptomatology of focal lesions situated in the neighbourhood of the Rolandic fissure. I have devoted special attention to the sensory disturbances and, with regard to motor symptoms, to the presence or absence of spastic phenomena. I have similarly analysed a number of cases with capsular lesions—twenty-six in all—but as our knowledge of these is far more complete than is the case with regard to the symptomatology of the cortical lesions, I will not burden this paper with an account of them; they are chiefly of interest for the sake of comparison.

In the first place I shall describe the methods employed for the study of sensory disturbances, which were on the whole similar to those used by Professor Petrén at this clinic. The notes of my cases, with special remarks, will follow, and I will then give a summary of the symptomatology of the sensory disturbances that result from cortical disease. The forms of sensation which I have investigated are tactile sensibility, the sense of superficial pain, the power of localization, and the muscular sense, especially the perception of passive movements. On the other hand, I have been compelled from practical reasons to neglect the sense of temperature, and I have not dealt with the more complicated functions such as stereognosis, &c. My observations were made on eighteen cases of cortical lesions, six of which have been examined post-mortem, and, in two of these, serial sections of those parts of the hemisphere which could come under consideration were studied microscopically. In the remaining twelve cases of monoplegia the diagnosis of a cortical lesion was not confirmed post-mortem, but I consider myself justified—with the support of Marie and Guilliain and others—in considering the localization of the lesion above the internal capsule sufficiently certain to draw conclusions with regard to the symptomatology of supra-capsular lesions. The study of my material has led me to conclude that certain differences in the disturbance of sensibility exist between cortical and capsular lesions, and that these differences occur so frequently that some practical importance can be attached to them.

I shall devote the next part of this paper to the question of the localization in the cortex of motility and the various sensory functions. In order to approach this question more fully I have tried to collect from the literature serviceable cases with an affection in the neighbourhood of the central convolutions which were confirmed by examination after death or during an operation, and cases of traumatic injury in which the site of the lesion could be safely assumed. With the support of these cases and the experimental physiological and histological brain-research of later years, I have tried to draw conclusions with regard to the cortical localization of sensation and motion.

On one point these researches permit us to draw a definite conclusion—viz., that the sensory and motor localization cannot coincide; one can conclude with certainty that motility is localized solely in front of the sulcus centralis and solely in the anterior central gyrus: whereas sensation, on the other hand, is localized solely behind the sulcus centralis, or more strictly to the gyrus centralis posterior, and at least to a part of the parietal lobe. I have not been able to arrive at any further definite conclusion as to the localization of sensibility, neither with regard to the extent of the sensory projectory area, nor to the localization within the same of the various sensory functions.

The last chapter of my paper deals with certain motor disturbances, especially the condition of the reflexes, in connexion with cortical lesions Marked increase of the reflexes, as well as the typical late contracture, have been absent in my eighteen cases of cortical lesion, in spite of the fact that the lesions in several of the cases were so old at the time of observation that a contracture ought to have set in if the cases were in this respect like the capsular ones. This important difference between capsular and cortical lesions does not seem to have received notice in the literature of recent years, though, as a matter of fact, this point did not escape two French clinicians of the earlier half of the eighteenth century, Rostan and Durand-Fardel.

The cases on which the following investigation is based have been to a large extent (viz., Cases I., V. to VIII., X., XI., XIII., XV., XVII. to XXIII.) observed at the medical clinic of the Academical Hospital of Upsala, and I am indebted to Professor Petrén for the large material which in this way has been at my disposal. Through the kindness of

Dr Wilkins, head physician of the medical part of the workhouse hospital in Stockholm, I had the opportunity of examining a number of cases of hemi- and monoplegia. Several of the cases examined there are recorded here, viz., Cases II. to IV., IX., and XIV. Cases XII. and XVI. were seen in private practice.

In the following pages the Roman numerals (X., &c.) indicate references to my own cases, and the ordinary numerals references to the Bibliography.

CHAPTER II.—METHODS.

I need not dwell upon the examination of motility, as this essay is chiefly concerned with the study of disturbances of certain sensory functions.

The functions which have been made the subject of a closer study are the senses of pain and touch, the power of localization, the perception of passive movements, and lastly, in some cases, orientation in space.

Tactile sensation has been tested partly by finger-touch and partly by cotton-wool touch, the latter method being very suitable for the detection of a slight degree of impairment, as has been pointed out by Fr. Müller. It must be remarked, however, that old people and persons with a rough skin may be unable to appreciate a cotton-wool touch on either side of the terminal phalanges, and that this "impairment" of sensibility may extend over the greater part of the palmar surface of the hand. With this very simple method one is able to obtain some idea of the intensity of the disturbance present; but, unfortunately, this cannot be expressed in numbers. No quantitative method of testing tactile sensation suitable for clinical purposes is mentioned in the literature.

My investigation of the sense of pain has been limited to superficial pain sensation. This was at first tested in the usual manner by letting the patient say whether he was being touched with the head or the point of a pin, but I soon found that this method was not sufficiently accurate to exclude minute disturbances. I have consequently tested, as far as possible, the superficial pain sense quantitatively.

Methods for the quantitative examination of the sense of pain have been invented by several investigators, such as Björnström, Mocutchowsky, Bechterew, Thunberg, and others. The instruments I have used are an algesimeter constructed by Thunberg, and the modification of the same by Alrutz. In the case of Thunberg's algesimeter the stimulus is applied by means of a pointed needle, *A*, (fig. 1), which is placed at *right angles* to the skin, whilst the part to be tested lies strictly in *the horizontal plane.* The needle is suspended from a light lever, *B*, which can be accurately equilibrated by means of a counter-weight, *C*. By placing weights at different distances from the needle, the pressure exerted can be varied. The lever is supported from beneath by means of an arm, *H*, attached to the handle *F*, and in its lower part bent at a right angle, and is thus prevented from sinking when the weights are put on.

The effect of the stimulus exerted by the needle is influenced not only by the weight but also by the nature of the point. On the other hand, the velocity with which the needle is applied is of no consequence when using Thunberg's instrument. The blunter the needle, the greater must be the weight to reach the liminal intensity of painful stimulation. The sensation of touch following a minimal stimulation of the pain sense is stronger when using a coarser needle, whereas the itching sensation that comes on later is more marked when finer needles are used. When the area of the point measures 0·001 – 0·0001 mm², the liminal intensity of stimulation remains nearly constant, and as the usual sharp-pointed needles to be had in the market have points of this size, *the effect of the stimulation depends exclusively on the weight.* The method is therefore based on measurement of the pressure needed.

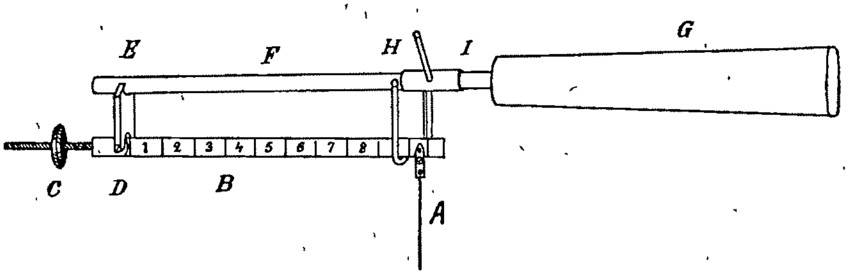

FIG. 1.

By means of Thunberg's algesimeter the liminal intensity may be determined with the greatest accuracy on all those parts of the body where the size and form of the instrument, which is unquestionably somewhat inconvenient, does not prevent is use. The necessity of placing the instrument at right angles to the horizontal part considerably hampers its employment in topographical determination of sensibility to pain, especially in patients affected with contractures.

These difficulties are avoided by using Alrutz's modification of the apparatus. In this algesimeter (fig. 2) the upper end of the needle is attached to a coiled spring, which is drawn out when the needle is pressed against the skin. By setting the spring, the strength of the stimulus can be varied; this is effected by means of a screw. On the scale, the weight acting on the needle is indicated, expressed in grammes. The area covered by the point of the needle is 0·001 mm^2, and the pressure exerted on the needle may be varied between 2-6 grm. in the usual instrument. The chief advantage offered by this apparatus is the independence it gives with regard to the position of the surface of the skin to be examined. On the other hand, the determination of the liminal intensity of stimulation is less exact than when using the apparatus of Thunberg, and the quickness with which the needle is applied to the skin influences the result obtained with the algesimeter of Alrutz. Further, it has seemed to me advisable to reduce the minimum pressure, a weight of 2 grm. being excessive when testing certain parts of the skin, such as that of the face, and especially the forehead, rendering it impossible to demonstrate in these parts a minute alteration of the physiological liminal value. In spite of these objections to the algesimeter of Alrutz, it is superior for clinical use to that of Thunberg, and for that part of the body on which my investigations were especially made—viz., the upper extremity—it has proved very convenient.[1]

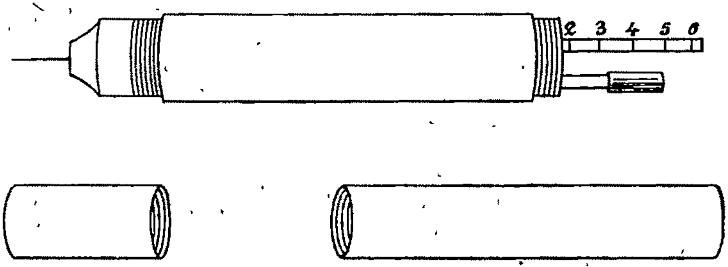

FIG. 2.

The liminal intensity of the stimulation of pain sensibility on different parts of the skin has been repeatedly the subject of investigation, especially from a clinical point of view. As, however, the methods employed are based upon different principles, it is evident that the results obtained must differ greatly. On this account I append a diagram (fig. 3) showing the result of an investigation with Alrutz's algesimeter on a healthy person—a labourer. As will be seen, a weight of 2 grm. is felt everywhere on the anterior surface of the trunk, as well as on the head and neck, whereas on the posterior aspect of the trunk areas are to be found showing a higher liminal intensity. With regard to the upper extremity, 2 grm. are perceived everywhere, except around the olecranon and on the hand. On the palmar aspect not even 6 grm. are, as a rule, perceived. On the dorsum of the hand the individual

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[1] The algesimeters of Thunberg and Alrutz may be obtained from the instrument-maker R se, Upsala, the price being 25 and 18 Swedish crowns respectively.

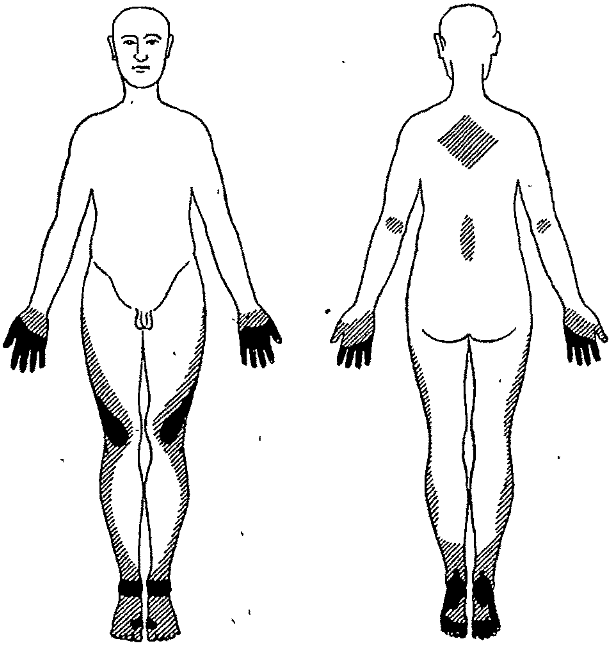
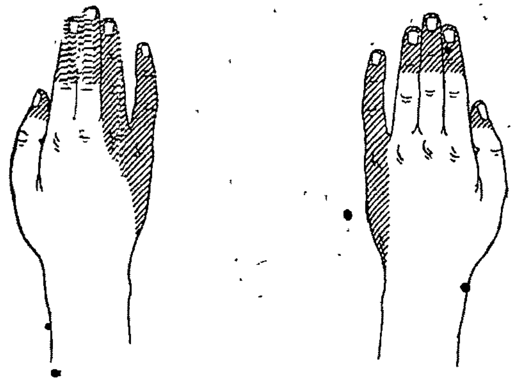


FIG. 3.

variations are very great; but I found constantly, as did Mocutchowsky with his method, that the liminal intensity was higher towards the ulnar side of the hand. This is of great importance when we have to determine whether impairment of painful sensations really does exist or not on any part of the hand. I append some diagrams of a number of similar investigations (fig. 4). On the dorsal and volar surfaces of the fingers the sense of pain was less sharp than on the surfaces opposed to each other according to the method I have used. On the lower extremity the liminal intensity varied a good deal, even



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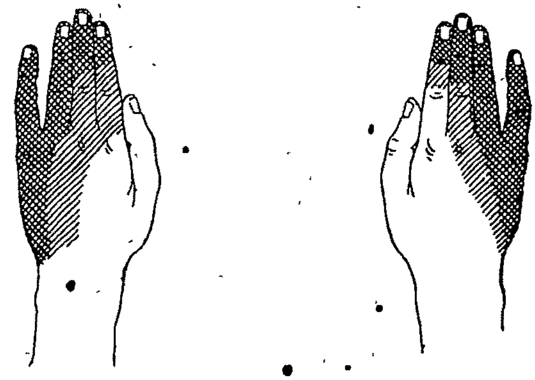

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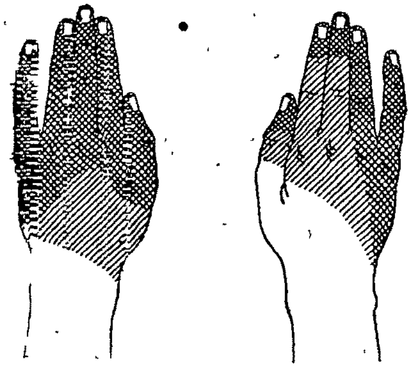
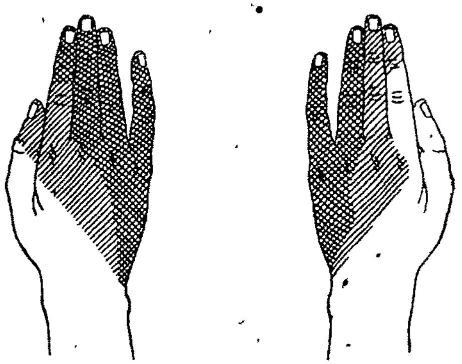
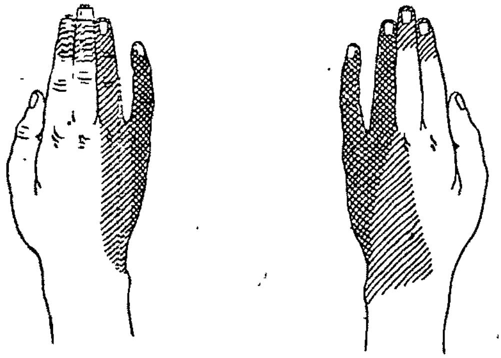


III

This patient has suffered from a compression neuritis of the left nervus radialis some years ago.



IV



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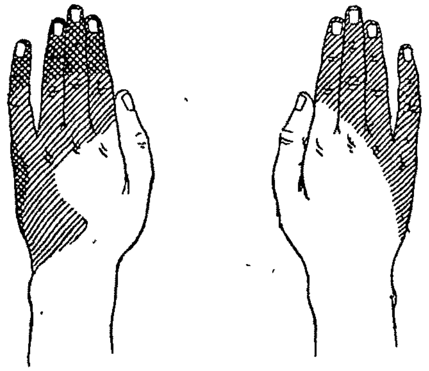

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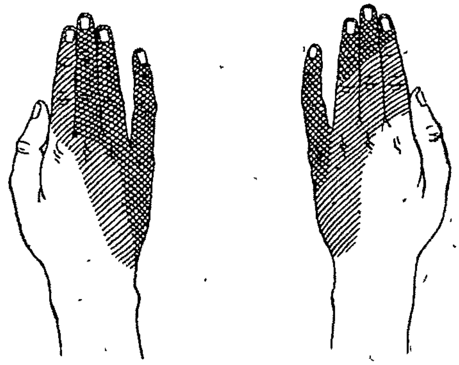

VII

in the more proximal parts. Specially noteworthy is the fact that on the outer surface of the thigh and over the patella the liminal intensity even exceeded 6 grm.; on the dorsal aspect of the foot 2 grm. was as a rule not perceived, but 6 grm. was distinctly appreciated, except over the dorsal and palmar surfaces of the toes and over the ankle-joint itself. On the sole of the foot 6 grm. was not perceived.

The sense of deep pain was not constantly examined in my cases; and the same applies to the sense of temperature, the fixation of the liminal intensity being impracticable on account of want of attention on the part of the patient.

The power of localizing touch and pain is of great interest. This function, which presumes of necessity the power of appreciating a touch as "a sensation of locality," does not exist alone, either under normal or pathological conditions; on the other hand, one may well have a sensation of being touched without being able to state exactly, or even approximately, the spot touched. As is well known, the power of localization varies greatly at different parts of the skin, more particularly according to the method by which it is investigated.

When the patient has to decide whether he is being touched with one or two points of Weber's compasses, the distance at which the points are felt double, when applied simultaneously, varies considerably in different parts of the skin and decreases distalwards on the upper extremity. If this power of discriminating whether one or two spots are touched is tested, not by simultaneous stimulation, but in such a way that the patient has to decide whether the touches coincide or not—*i.e.*, on successive stimulation—it is found to be greatest on the distal part of the upper extremity. The distance at which two points are felt double is smaller when this method is employed than with simultaneous stimulation. Simultaneous stimulation has been most employed in my investigations.

This method has reference only to the estimation of two touches in relation to each other; the absolute power of localization, on the other hand, was tested by asking the patient to name the place where he has been touched when his eyes are closed, only requiring him to indicate it on a diagram; or a second or two after the touch he was allowed to open his eyes and point to the spot. The error is often greatest by the first method. In this work the investigation has almost entirely been carried out in the latter way. The errors of the method here employed under normal conditions on the upper extremity are, according to Brugsch and Schittenhelm, as follows:—

Finger (vola)	0·1—0·15 cm.
Finger (dorsum)	0·2 cm.
Hand (vola)	0·5—0·7 cm.
Hand (dorsum)	0·5—0·8 cm.
Forearm	0·6—0·9 cm.
Upper arm	1·4 cm.

I have tried to study the so-called muscular sense more closely in my cases, thereby paying attention almost exclusively to one of the functions generally included under the term muscular sense—viz., the perception of passive movements. I do not intend to deal with the physiological substratum on which this function is based, nor of its relation to other functions of the muscular sense, such as active movement, appreciation of position, sense of strength, &c. One might, however, object that as the appreciation of passive movement has rarely attracted the attention of the patient, its study would be on that account of no importance under pathological conditions; but the experience from tabes has shown that a close relation exists between disturbances of this function and of co-ordination—a fact which proves its value as a clinical method of investigation. Its study has one advantage in that it can be tested as soon after a hemiplegia as the psychical condition of the patient permits a detailed examination of sensibility, whereas the appreciation of active movements, the sense of strength, &c., demands the recovery of motility. From this point onwards the expression "muscular sense" is used in the sense of appreciation of passive movements.

The perception of passive movements depends partly on the angle, partly on the rate of the movement; but with moderate differences in rate the latter factor becomes of subordinate importance (Goldscheider). Under normal conditions the smallest movement the direction of which can be recognized is, according to Goldscheider, in the inter-phalangeal joints 0·7°—1·8°, in the metacarpo-phalangeal joint 0·4°—0·5°, in the wrist-joint 0·3°—0·6°, in the elbow-joint 0·5°—0·8°, and in the shoulder-joint 0·3°—0·6°. In order to measure the size of the angle necessary for the perception of the movements, Goldscheider has constructed an apparatus which can be adapted to most of the joints. During the course of my investigations I found, however, that even without this apparatus one is able to get very near the physiological minimum perceptible in a number of joints by the aid of the simple method of investigation generally employed at this clinic—viz., by performing small, regular movements after having taken a firm grip of the peripheral part of the respective extremity, avoiding stretching the skin, and asking the patient to state the smallest movement perceptible and its direction. As the distance between the metacarpo-phalangeal joint and the part of the terminal phalanx which may be conveniently grasped is about 70—90 mm., the range of movement at this joint, when the terminal phalanx is moved 1 mm., is about 0·6°—0·8°, an angle which comes very close to the normal minimum of 0·4°—0·5°. To make a movement of only 1 mm. is, after some practice, not attended with any difficulty. When we have to deal with the larger joints it becomes, on account of the greater length of the lever, still easier to get down to the minimum perceptible. I did not, however, succeed in this at the inter-phalangeal joints. From this it follows that, in the metacarpo-phalangeal and the more proximally situated joints, minimal alterations in the perception of passive movements can be demonstrated, and that *the absence of impairment demonstrable by this method entitles us to presume that the patient appreciates the normal minimum perceptible.*

In the case of greater impairment of the perception of passive movement I have always tried where possible to obtain an approximate value of the minimum perceptible by measuring the length of the lever and the distance which its distal part has to be moved in order to cause the movement in the joints of the wrist, elbow, and shoulder to be appreciated. I have, as a rule, taken the mean of ten such measurements, or, when the difference between the different measurements has been greater, the mean of twenty.

The "appreciation of position" is generally tested by making the patient imitate with the non-paretic hand the passive movement of the paretic one, or else he was made to move the non-paretic hand towards the paretic one, this latter occupying different positions. In my experience the former method is not sufficiently delicate to detect minor alterations in the appreciation of position, and it often happened that I was able to detect such alterations only by the second method, especially when this was applied in the modification employed by Horsley and Slinger—viz., by letting the patient point at a glass slide divided into centimetre squares with the index-finger of one hand and move the other hand towards the pointing finger. It deserves mention, however, that in both these tests we are dealing with a very complicated function, as both the sensibility and motility of the non-paretic, as well as the paretic, side must influence the result; it is therefore unjustifiable to compare the results obtained by these methods with those obtained on examining the muscular sense.

Horsley and Slinger varied this method in different ways when investigating the "orientation in space" under normal conditions. They found that our ability to estimate the position of the hand varies considerably, according to its position, in relation to the body; that it is greater the nearer the hand is to the centre of gravity of the body, and then decreases as the position of the hand is further upwards or outwards. Further, the precision is greater with the arm flexed than extended. In the mesial xyphoid plane the error is generally 1·25—1·5 cm. when the fingers are at a distance from the trunk corresponding to the wrist-joint. The most common form of error is in the proximal direction. In Horsley and Slinger's investigation this occurred about three times to every twice that the error occurred in a distal direction (this form was characteristic for the overhead position).

In my own investigations I have naturally not been able to vary this method in the same manner as the above-mentioned authorities; but I have had to confine myself to making the patient move the non-paretic hand towards the paretic one at different heights of the mesial plane, the slide being held in the sagittal plane, and *vice versa* if sufficient motility was present.

This investigation consequently refers to the orientation in space of the hands. If we may now assume that the non-monoplegic arm is in possession of normal orientation both at rest and in motion, then this method admits of a comparison between the orientation at rest and locomotion of the monoplegic arm when the patient is made to perform these movements alternately with both arms.

In order to try the orientation of the arm when moving, I have also made use of the method described by Blix in the year 1883. The patient is placed in an easy position—e.g., sitting on a chair with his back resting against the back of the chair, or standing with the back firmly supported. The arm hangs vertically by his side, and he holds a pencil in his hand. At a distance of an arm's length in front of him and at a level with his shoulder a paper is fastened to a vertical board or screen, and the patient is required to raise his arm, without otherwise moving, and touch with the point of the pencil a certain spot previously marked out on the paper, and then move the arm back to the starting position. Then he is asked to repeat this test with closed eyes. This experiment is repeated again and again, the patient performing the movements alternately under the control of the eyes and without their aid. Each time the pencil makes a mark on the paper at the end of the experiment, the distances between the fixed point and the different marks are measured with a scale and the sum divided by the number of trials.

Öhrvall, who has employed this method for an investigation on exercise and fatigue, found it extremely useful. But as an evident and early effect of exercise and fatigue is not desirable, this makes it less suitable as a clinical method of examination. Besides he points out that it is not only the orientation of the arm which in this way is tested, as the orientation capacity of the eyes is also utilized. As it is impossible to eliminate this factor in a clinical investigation, it is a serious drawback when, as is the case in this work, one has to compare the two sides.

The difference between this method and the one indicated by Horsley and Slinger is evident. In the one case it is the sense of position of the recorded side, in the other the orientation of the visual sense that influences the result, and in both cases in addition the orientation of recording hand.

However, a severe paresis interfered so much with this investigation in the greater number of cases that I was unable to make use of the method of Blix, and I can consequently refer only to two cases, where the paresis was moderate, in which this method was used. In cases of paresis Horsley and Slinger's method is easier to use than the one indicated by Blix.

Finally, the stereognostic power has been tested in the usual manner, but in detail only, in those cases where impairment of the more elementary functions could be eliminated.

CHAPTER III.—CASES.

§ 1.—*Capsular Lesions* (*Cases I. to V.*).

I have examined twenty-six cases of hemiplegia with regard to sensory disturbances and spastic symptoms, increase of reflexes and contractures.

A high degree of reflex exaggeration and contractures of the arm as well as of the leg were found in all these cases, with the exception of Case V., which is described below, in which spastic symptoms were present only in the leg. Sensory disturbances, on the other hand, were present in nineteen cases of hemiplegia (of which eight were verified post-mortem) in spite of the fact that the intensity of the paralysis varied considerably in different cases, and in these nineteen cases sensibility to touch and pain as well as the muscular sense were affected. In the remaining seven cases I was unable to decide whether sensory disturbances were present or not on account of the psychical condition of the patient.

CASE I.—J. L. B., aged 66, merchant.

Considerable alcoholic abuse in the past history. On March 6, 1907, quite well, but next day, immediately after getting up, he noticed that the right leg would not support him; paræsthesiæ of the right side and arm developed, the right limbs became paralytic after the lapse of one hour, and there was difficulty in speaking. The two sides of the face were unequal.

On March 7.—Wandering, aphasic. Facial paresis, right arm and leg flaccid; hemi-analgesia, reflexes of the right side absent. Arterio-sclerosis.

March 8.—Still wandering in mind, but reacting on being addressed. No hemianopsia demonstrable; complete paralysis of arm and leg; considerable degree of paresis of the right facial and hypoglossal nerves. Complete hemianalgesia as before. and marked hypalgesia also of the left lower extremity.

March 11.—Understands spoken words perfectly. Paralysis as before. The analgesia is found to extend to the "healthy" side, at least on the trunk, for a distance of about 2 cm. Total disorientation with regard to the position of the arm and leg.

March 21.—The analgesia of the right side of the face diminished, but distinct impairment still present laterally; on the trunk deep, pricks are appreciated near the middle line but not laterally; the analgesia of the arm and leg still complete. Hypalgesia of the left leg and foot remains. Patellar reflex increased; Babinski's sign on the right side. Triceps and biceps reflexes still absent.

April 1.—Paresis of the face and tongue diminished. Paralysis of arm and lower extremity. Appreciates deeper pricks all over the face and trunk, but less distinctly laterally; on the right upper arm and thigh deeper pricks are also felt. No hypalgesia now to be found on the right leg. Tactile sensibility and sense of pressure absent on the right side. Inability to localize pain in arm and leg, localization fairly good on the trunk. No appreciation of passive movements of the right arm and leg. Is able to say solitary words, but the speech is blurred and difficult to understand. Can read vertical letters.

Discharged on April 4.—After his return home the speech improved greatly; at the end of about one month the patient was able to talk pretty freely although still rather slowly. The paralysis remained, but sensibility gradually returned.

July 22, 1908.—Speech still a little slow and somewhat blurred, but the patient has not to search for the words. No paresis of the face or tongue. Rotation outwards, and adduction of the shoulder-joint can be performed with pretty good strength, and the same applies to the raising of the shoulder; other movements almost lost. Flexion of the elbow-joint pretty good; extension much weaker. Paralysis of the hand and fingers. No contracture of the shoulder-joint; slight contracture of the elbow-joint in the flexed position; pronounced flexion contracture of the fingers (during several months he was treated daily with passive movements). Flexion of the hip and knee pretty strong. Considerable contracture of the ankle-joint. Reflexes of the right arm and leg greatly increased. Ankle clonus. Babinski's sign.

Appreciates a cotton-wool-touch everywhere on the arm except on the distal or the two distal phalanges, where, however, a finger-touch is appreciated. On the leg and foot cotton-wool is appreciated everywhere, but with uncertainty on the toes; it is the same on the other side. Is able to differentiate everywhere between head and point of a pin. The algesimeter of Alrutz reveals a reduction on the fingers, less than 4 or 6 grm. not being felt on the distal phalanges, and even this latter weight vaguely. On the healthy side 2-4 grm. are everywhere appreciated without hesitation. The after-sensations following deep pricks last longer and are more uncomfortable on the right hand than on the left, but one cannot speak of any real increase of painful sensation.

Alrutz 4 is, as a rule, appreciated only in the upper part of the leg. On the foot the diminution is still greater. The patient differentiates well between touch and pressure on the right arm and leg. Appreciation of the vibration of a tuning-fork is equal on both sides. Passive movements, even minute ones, are appreciated in the shoulder-joint, but their direction is not accurately recognized until the movement has reached about 5° or more. At the elbow-joint movements of extension are appreciated, even minute movement, but flexion not until about 10°. He records the direction of movements of extension correctly at about 10°—15°, and flexion at about 15°—25°, regardless of the starting positions. At the wrist and finger-joints movements are not appreciated until the soft parts are considerably stretched, and even then the direction is vaguely described.

At the hip- and knee-joints even small movements are exactly appreciated. At the ankle-joint contracture does not permit more than a small range of movement. These are not appreciated. Large movements of the toes are not appreciated on the right side, but on the left side even slight movement is perceived.

Localization of touch is very uncertain on the hand and forearm, and without any regularity as regards atopognosis. On the upper arm it is fairly certain.

There is total disorientation of the position of the right arm, he finds the hand only by feeling his way. At each trial he regularly moves the left hand towards the place which has been occupied by the right one at the preceding trial (perseverance of visual impression).

The diagnosis of a capsular lesion in this case needs no further comment, considering the total and lasting hemiplegia and the rapid recovery of the aphasia present. It is not the character of the hemiplegia which is of interest for my investigations, but two remarkable symptoms presented by the patient—viz., the hypæsthesia present also on the "healthy" side of the trunk and lower extremity, and the fact that the disturbance of muscular sense at the elbow-joint was greater in flexion than in extension. I shall return to these questions when reviewing in brief the symptomatology of the sensory disturbances.

I record here three other cases of capsular lesion with post-mortem examination; in the first of these the lesion also involved the thalamus opticus. I also record a case in which the projection tracts were involved, not in the capsule itself, but immediately above it. All these four cases presented definite motor as well as sensory disturbances, and in Case II., in which the thalamus was involved, this sensory disturbance was especially intense. Case II. showed also slight signs of hypæsthesia in the lower extremity of the sound side. In the other cases it was evident that the "simple" cutaneous senses were those least affected, a fact to which I shall return in connexion with the sensory disturbances due to cortical lesions.

Case II.—K. K., aged 74, had an apoplectic stroke in September, 1906, and since then weakness of the left side.

January 22, 1907.—A considerable paresis of the left lower, slight paresis of the left upper face; the tongue deviates towards the left; paralysis of the left arm, considerable paresis of the left leg; nearly complete contracture of the arm in the flexed position; contracture of the ankle-joint in the plantar-flexed position.

Does not feel a cotton-wool-touch on the left side of the face, does not feel a hard touch or pin-prick outside a sagittal plane, passing through the outer orbital angle. On the trunk a cotton-wool-touch is felt 2 to 3 cm to the left of the middle line. On the lateral parts of the trunk, as well as on the extremities, no appreciation of a heavy touch nor of pain.

No knowledge of position nor of appreciation of passive movements on the left side. On the right side a cotton-wool-touch is not felt below the knee nor on the two distal phalanges of the fingers. A somewhat harder touch and a prick are appreciated and correctly localized on the right side, a certain degree of uncertainty as regards minimal movements of the fingers.

Death on March 19, 1907.

Post-mortem.—An extensive old lesion occupying almost the whole of the internal capsule, the greater part of nucleus lentiformis, and the external and lower portions of the optic thalamus.

Case III.—K. V., aged 59, apoplexy in the middle of May, 1904; left-sided hemiplegia, with increased reflexes and contractures.

Enlargement of the heart towards right and left, presystolic and systolic murmurs and a thrill.

January 20, 1907.—Left hemianopsia: movements of the eyes unaffected. Painful and tactile sensibility normal on the area supplied by the trigeminal. Paresis of the lower part of face. No deviation of the tongue. Almost complete paralysis of the left upper extremity, only a slight degree of the mobility of the shoulder persisting. Passive movements of the shoulder-joint restricted.

At the elbow-joint contracture to nearly a right angle in the flexed position. Passive movements can be performed between 45° and 90°. Pronation-contracture and moderate flexion-contracture of hand and fingers. Passive mobility of the wrist 45° palmarwards to 45° dorsalwards. Marked paresis of all the joints of the lower extremity, especially of the distal. Very marked contracture of the hip- and knee-joints in the flexed position, plantar contracture of the ankle-joint.

No appreciation of cotton-wool on the hand and fingers, but of finger-touch everywhere on the left upper extremity. Sense of pain (pin-prick) somewhat uncertain distally. The sense of deep pressure unaffected. Localization (successive stimulation) bad in hand and forearm. In the fingers and hands a considerable proximal reference; in the two ulnar fingers, possibly also in the index-finger, atopognosis in the direction of the middle finger. No alteration of painful and tactile sensibility in the lower extremity, localization good judged by verbal statements.

Appreciates correctly even minimal movements of the shoulder-joint, and at the elbow-joint movements of 5° to 10°. At the joints of the hand and fingers movements of 30° are not appreciated, and considerable degree of uncertainty is also shown in larger movements. No perception of passive movements at the joints of the toe nor at the ankle-joint of those small movements which the contracture permits; at the knee- and hip-joints exact perception of minimal movements.

Death on June 2, 1907.

Post-mortem.—Almost the whole of the caudate nucleus, the whole of the lenticular nucleus as well as internal capsule in its whole extent are occupied by a large apoplectic cyst. The optic thalamus was not directly involved macroscopically, but on the right side was atrophic and indistinctly marked.

CASE IV.—K. V. P., aged 52. On June 1, 1896, an apoplectic stroke followed by total hemiplegia and marked spasm.

January 17, 1907.—Speech slow and somewhat blurred; no aphasia. A slight paresis of the upper and lower half of the face on the right side; soft palate unaffected; difficulty of swallowing; the position of the tongue normal.

Severe paresis of the right arm. Active mobility of the shoulder-joint through about 45°, the power being pretty good. Passive movements only slightly better; arthritis. Active movements of the elbow-joint of nearly full excursion, but the power greatly reduced. At the wrist-joint palmar flexion to almost right angle and extension normal, but the strength extremely poor. Dorsal flexion cannot be performed actively. Flexion of the fingers remains, but extension is poor; the power very slight, it cannot be measured with the dynamometer. Severe paresis of the leg; active movements of the hip- and knee-joints can be performed to full excursion, complete plantar contracture of the ankle-joint.

Appreciates badly a cotton-wool-touch on the upper arm and thigh, not at all on the fore-arm. Appreciates vaguely finger-touch on the hand, as well as on the foot and leg below the knee. No appreciable reduction of painful sensibility. Appreciates also a deep pressure as such on the hand.

Localization (successive stimulation) bad on the hand; proximal atopognosis of 5 to 8 cm., greatest at the ulnar side; in addition a trace of faulty localization towards the axis of the middle finger, likewise greatest in the two ulnar fingers. Above the wrist the power of localization rapidly improves and becomes normal at about the middle of the fore-arm. Localization, when tested by the patient's own statements, showed much greater error, and no regularity with regard to the errors could be ascertained; localization tried by æsthesiometer was found reduced on the right hand; he appreciates only one point when touched on two fingers, on the upper arm, he was accurate as on the other side, but on the fore-arm somewhat less good. Localization on leg and foot pretty good, at least when compared with the other side. Appreciation of passive movements good at the shoulder-joint; at the elbow-joint the direction of movements of 15° are not appreciated with certainty, though 1° is recognized on the left side, but the patient recognizes that a movement takes place, even when this is very small.

At the wrist-joint a movement of 45° is recognized as such, but the appreciation of the direction is uncertain even of movements of largest possible range. No appreciation of passive movements of the fingers. At the hip all movements are correctly recognized, but at the knee uncertainty of the direction of movements of 4° to 8°. At the ankle-joint only movements of pronation and supination could be tested on account of the spasm; no appreciation of these movements.

Passive movements of the right hand are pretty well imitated by the healthy one, and at the elbow-joint the error of the angle is at any rate not greater than the angle necessary to recognize exactly a movement on the right side.

Reflexes of the right arm and leg lively; ankle clonus; moderate contracture of the arm, severe of the leg.

Post-mortem.—A large softening involving part of the lenticular nucleus and the upper portion of the internal and external capsule on the left side. Thalami macroscopically normal. Small cysts were also found in great number in the anterior part of the internal capsule of the right side, but without any softening. The basal arteries had thickened walls

CASE V.—J. S. L., aged 13. At seven years of age rheumatic fever. March, 1907, polyarthritis, which improved under treatment with salicylates; organic heart disease diagnosed on this occasion. On March 16 the patient was again taken ill with headache and vomiting, which got still worse on the 20th, when, in addition, a left-sided hemiplegia developed. The patient remembers quite well that the sensibility of the whole of the left side was at first greatly impaired. At the end of about one month sensibility of trunk and leg good, that of the arm still bad. The paralysis has remained almost unaltered until the last month, when the patient began to make small movements of the fingers and toes. Admitted July 15.

September 2.—The patient is pale, thin, with reduced muscles. Mitral insufficiency. Induration of the right apex. The patient is well aware of his illness, is attentive, but gets easily tired. No hemianopsia. The left nasolabial fold somewhat less marked than the right. The left half of the face partakes less in mimic and voluntary movements. Elevation of the shoulder weaker on the left side; severe paresis of the shoulder-joint. At the elbow-joint movements of about 20° can be performed, but with very little power. The same applies to the joints of the hand and fingers, except the thumb, which is paralysed. Passive movements of the hand and fingers normal; passive extension of the elbow-joint not complete; passive mobility of the shoulder-joint about 60°. Almost complete paralysis of the knee-, ankle-, and toe-joints. Movements of the hip somewhat better.

In the area supplied by the trigeminal nerve no impairment of tactile or painful sensibility (Alrutz). On the radial half of the hand and on the fingers cotton-wool is not appreciated, but a finger-touch is; otherwise no disorder of tactile sensation in the hand. The sense of pain, as tested by pin-prick, showed no evident impairment. With Alrutz's algesimeter, a weight of 2 grm., a prick was not felt below the elbow, the limits of the sensory loss for 4 and 6 grm. coinciding very nearly with those obtained with cotton-wool. On the upper arm and trunk no objective alteration of the senses of touch and pain, but the patient asserts there is much greater pain after deep pricks on the left side. On the lower extremities a cotton-wool-touch is felt everywhere except on the toes. Does not certainly feel a prick with 2 grm. on either foot, 4 grm. generally with certainty. Greater pain on the left side after pin-pricks.

Power of localization in the left arm above the wrist normal; below the wrist a touch is generally localized towards the wrist and with marked predilection towards the ulnar side. In the lower extremity the power of localization, tested by verbal statements, is good except in the toes.

Tactile discrimination (Weber's compass): On the left hand and fingers two points are not felt until at a distance of 5 cm.; on the right side normal values. On the arm two points are not even discriminated at a distance of 8—10 cm. On the chest two points are felt at a distance of 2·5—3 cm. on the right side, 5 cm. on the left, on the abdomen at a distance of 3—4 and 6—7 c.m. respectively. On the left lower extremity 7—8 cm. are necessary for the discrimination of two points. On the right leg normal values.

Appreciates movements of the left shoulder-joint of about 5°, and recognizes their direction correctly at about 15°; at the left elbow-joint movements of 10°—15° are perceived, and their direction clearly recognized at 45°, but he is uncertain at 30°. At the wrist-joint only extreme movements are perceived, but these are correctly described. Extreme movements of the little and ring fingers are likewise appreciated, but not those of the other fingers. At the hip- and knee-joints minimal movements are correctly estimated, at the ankle-joint only large ones (45°), in the toes only extreme ones. At the right ankle-joint even minimal movements are correctly recorded, but in the toes there is uncertainty with regard to small movements.

Estimation of heavy pressure and vibration showed no evident difference on the two sides, but there was a diminution of the power to localize these impressions. Biceps- and triceps-reflexes absent on the left side, knee-jerk distinctly increased. Babinsky and slight ankle-clonus on the left side. No contracture of the joints of the hand or fingers. Slight contracture in the flexed position of the left elbow-joint. Moderate contracture in the plantar flexed position of the ankle-joint on the same side. On September 20 and 26 renewed examinations, with the same result except that the perception of passive movements of the right ankle-joint was somewhat less accurate than before.

Death on November 28.

Post-mortem.—The soft membranes thickened and adherent to the brain. Marked injection, especially over the lower portion of the central convolution of the right side. The surface was otherwise unaffected. Thrombosis of the right arteria fossæ Sylvii. On frontal sections there was found in the right hemisphere immediately underneath the cortex of the posterior part of the frontal convolutions and the lower half of both central convolutions a well-defined structure of darkish-brown colour and firm consistency, surrounded by a narrow, blood-stained zone, outside this the brain-substance showed a yellowish discoloration and signs of breaking down over an area varying in size from a few millimetres to 1 cm. The lesion lay between the internal capsule and the cortex of the lower half of the central convolutions immediately above the lenticular nucleus, the outermost segments of which were partly softened; posteriorly the softening extended under the cortex of the parietal lobe. The lesion affected to a very great extent the projection fibres of the central convolutions, only a small portion of those nearest the ventricle were free from microscopical signs of softening. Cortex and superficial layers of the subcortex of the upper third of the central convolutions appeared intact to the naked eye.

Thus, in this the fifth case the lesion had destroyed both the cortical centre and the projectory tracts for the arm, but only the projectory tracts of the leg. Considering the extensive and intense destruction within the centre for the arm, I did not expect to find such a marked difference with regard to motion and sensation at the ulnar and radial side of the hand, a condition which probably can be explained only by assuming that it was not the internal capsule itself, where the tracts become mixed with each other, that was affected by the lesion.

The fact that no spastic phenomena were present in that extremity, the cortical centre of which was destroyed (viz., the arm), was of special interest; on the other hand, the leg was spastic, and, though its projectory tracts had been injured, its cortical centre was unaffected.

§ 2.—*Cortical Lesions (Cases VI. to XXII.)*

(*a*) *Pure Motor Symptoms (Cases VI. to XII.)*

CASE VI.—J. H., aged 44, a farmer.

Taken ill in the beginning of September, 1906, with shivering and pain in the chest, since then in bed. About one week later difficulty in speaking ("stiffness of the tongue") and right-sided paresis.

On admission, September 19: General condition very bad; pale and cyanotic; temperature 39·4 C., pulse 102; signs of right-sided pneumonia.

Myosis on the right side. Speech blurred and slow. Incomplete hemiplegia. Dynamometer left hand 25, right hand 15. No disturbance of cutaneous sensation or of muscular sense. Knee-jerk increased on the right side. Reflexes otherwise normal. No Babinski.

September 22.—No facial paresis; the motility of the leg normal; diminution of the paresis in the arm. Dynamometer right 16, left 20. The strength of the wrist-movements somewhat less on the right side. The difference is more marked at the elbow, where the power of extension is considerably reduced. The shoulder-joints also present some difference on the two sides, raising of the arm and abduction being weaker on the right. The movements of the scapula itself practically normal.

Later there were frequent and severe rigors with unaltered physical signs. As nothing abnormal was detected in the abdomen, and there was no sign of any metastatic process, it was considered probable that an abscess was developing in the lung. An increasing dulness over the right lung and a rapid change for the worse in the general condition of the patient were the first signs of an empyema, which it seemed probable was perforative. The patient was transferred to the surgical ward. An operation showed a perforation of the lung and a communication between the empyema and an intrapulmonary cavity. Death occurred on October 2.

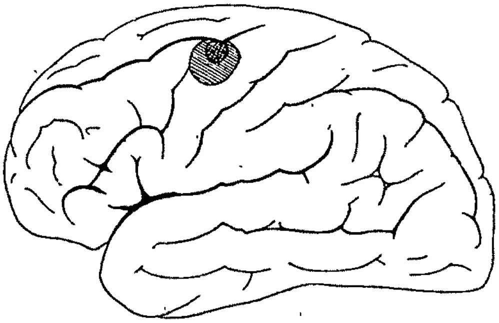

FIG. 5.

Post-mortem.—Two empyema cavities with well-defined walls communicating with a cavity in the lower lobe of the lung. Two other abscesses of the lung.

The brain: Over the middle third of gyrus centralis anterior and the adjacent part of gyrus frontalis medius the pia mater showed considerable injection over an area of about 2 cm. diameter and with diffuse borders. Corresponding to this area the brain-substance was somewhat prominent without any difference in its consistency. The hardened brain showed on frontal sections a distinct hyperæmia of the cortex over this area.

Microscopically, obvious changes were to be found within this part—viz.

considerable hyperæmia of the pia mater and cortex, and the most superficial part of the subcortex. Over an area the size of ½ cm. at the posterior part of sulcus frontalis superior the pia mater was infiltrated with round cells, especially in the neighbourhood of the vessels. Outside of this area small groups of round cells were seen around the vessels. In the most superficial part of the subcortex an early softening was visible, but otherwise hyperæmia only and proliferation of the endothelium. Outside the area of macroscopical change nothing abnormal could be seen by the microscope.

In the case I noted a pure motor paralysis without any sensory symptoms (at first hemiplegia, then monoplegia), due to an affection in the anterior central convolution. Further, it must be observed that the reflexes in the arm—*i.e.*, the extremity which was the most affected—were normal, while the reflexes of the leg, which presented motor symptoms only in the beginning, were increased.

In this case the cortical localization was confirmed by post-mortem examination. This was not possible in the great majority of the following cases. It may appear rash to conclude, as I have done, that the symptoms were due to cortical lesions in these cases in which the diagnosis has not been confirmed post-mortem. Horsley and Beevor found in *Macacus simius* that the motor bundles for different parts of the body separated from one another within the genu and posterior limb of the internal capsule. This fact has been generally applied to human pathology—for instance, by Monakow [141] and Dejerine [48]. Monoplegia might consequently result from a capsular lesion. But on considering the extensive area occupied by the posterior limb of the internal capsule, and the not infrequent occurrence of small lesions in these parts, capsular monoplegia ought not to be rare. But, as pointed out by Dejerine himself, limited lesions in this part rarely give rise to monoplegia, but, as a rule, produce hemiplegia. With the support of a large material Marie and Guillain hold that even a small lesion in the internal capsule is always followed by hemiplegia, never by monoplegia. Roussy found the ame (in his cases 1—3): the paralysis, though minimal, was of hemiplegic type, as a result of small lesions in the posterior limb of the internal capsule. Marie and Guillain explain this fact by assuming the subdivision of the motor bundles found in *Macacus simius*—and also in he dog (Franck and Pitres)—does not exist in man. A limited lesion of the posterior limb of the internal capsule produces a diffuse degeneration of the pyramidal tract within the medulla oblongata and the spinal cord, and a limited lesion of the motor zone causes a much more extensive degeneration of the internal capsule than one would expect according to the common scheme. A case of Bikeles, published last year, supports this view. In this case a lesion was found which had left the uppermost third of the motor zone completely unaffected. With Marchi's method a degeneration of relatively moderate intensity was found throughout the whole motor part of the internal capsule, in spite of the fact that sections from the medulla showed that the pyramidal fibres of the leg were not affected. Mellus (cit. Bikeles) also found that the different bundles of the pyramidal tract were mixed with each other in the internal capsule. And it seems to me that Long's [119] two cases of old monoplegia certainly point in the same direction. In one of these the lesion was situated in the centre for the leg, and with Weigert-Pal's staining, degenerated bundles could be followed to the foot of the corona radiata, whereas not a single distinctly degenerated bundle was observed within the posterior limb of the internal capsule, but only a slight diminution of the mass of the projection tracts. Long's second case was a porencephalitic (intra-uterine ?) cyst in the lower two-thirds of the motor zone, and in this case, too, there was no circumscribed atrophy or sclerosis in the internal capsule, but only a marked diffuse atrophy of the same. Long's statement that he has seen a case of capsular monoplegia—evidently Case 12 in his thesis [118]—is not altogether free from objection, as the patient had, at least subjectively, a *sensation de faiblesse*.

From these facts one seems entitled to conclude that the lesion in the case of a monoplegia is situated not in the capsule but in the cortex. On the other hand, it goes without saying that an extensive cortical lesion, as is well known, may give rise to a hemiplegia without the capsule necessarily being directly affected.

CASE VII.—A. J. A., aged 68, farmer. Notes by Mr. Ahlberg. (Abstract.)

For the last twenty years the patient has suffered from shortness of breath and palpitation of the heart, accompanied lately by cough. On December 27 the sleigh in which the patient was riding was upset, and he received a slight blow on the top of his head. He felt no discomfort, and put the sleigh right himself. About half an hour after the fall he felt the left arm getting weaker, and at the lapse of another half-hour the hand and fingers were completely paralysed; a few hours later the whole arm was useless. He was fully conscious the whole time, and felt no giddiness. A slight headache from which he had been suffering the whole day increased somewhat. Later in the evening he had some twitches in the paralysed arm. His condition remained unaltered during the next few days.

The patient was admitted into hospital on January 4, 1907.

Condition January 7-12.—Rather low. Badly nourished, with ill-developed muscles. Emphysema. Peripheral arterio-sclerosis. Blood pressure by the

Riva-Rocci-Landergren apparatus 150-160. Examination of the heart revealed aortic stenosis and mitral incompetency. The urine contained traces of albumin but no tube-casts.

Psychical conditions fair. The cranial nerves presented nothing abnormal.

Total paralysis of the left arm, muscular tension somewhat increased, especially in the flexors of the elbow. The motility of the right arm and the lower extremities normal.

No disorders of cutaneous sensibility nor of muscular sense could be ascertained. The patient localized his perceptions well and estimated correctly, even small movements of the joints. Triceps, patella and Achillis-tendon reflexes normal. Plantar reflex normal, abdominal and cremasteric reflexes absent on the left side.

On January 8 I had an opportunity of examining the patient. His intellect was clear, but he was somewhat inattentive. To cotton-wool there was no appreciable diminution of the tactile sense when compared with the symmetrical points on the other side. Sense of pain (pin-prick) normal. Power of localization normal.

Muscular sense, as far as I was able to make out, on account of the inattention of the patient; there was nowhere—not even in the finger-joints—any diminution of the perception of passive movements.

Passive movements of the left hand were correctly imitated by the right one.

Discharged January 18.

In this case the lesion developed after a trauma, but the slow appearance of the symptoms, without loss of consciousness as well as the slight trauma, must argue against the existence of a traumatic destruction of the brain-substance. On the contrary, the mode of onset of the paralysis is very characteristic for a softening, with its slow development, without any disturbance of the consciousness. This diagnosis is supported by the arterio-sclerosis of the patient. The lesion was probably cortical, considering the complete but isolated brachial monoplegia.

No examination was made earlier than a week after the accident, and then there was a complete absence of disturbance of cutaneous sensibility and of senses of position and of movement. I was not able to observe this case from the beginning, and I know nothing of the condition of sensation immediately after the accident. Nevertheless, this case proves that a complete motor paralysis may exist without any sensory disturbance, even so early after the appearance of a severe monoplegia. In this respect the following case is still more instructive.

I also observed in this case a slight rigidity in the flexor muscles of the elbow-joint—*i.e.*, a small trace of a "Frühkontraktur." The tendon reflexes were, however, quite normal, and Babinski's sign did not appear.

CASE VIII.—E. W., aged 58, business man.

Lues, 1879. Only one course of mercurial treatment (30 inunctions). Married 1891. The wife has had no miscarriages. Children healthy. Especially as a young man he drank spirits freely, and continued to drink during his later years. During the past year bladder trouble, with frequent straining and inability to commence the act of micturition. No signs of stricture or prostatitis. No cystitis, according to his medical attendant.

On March 1 at 12.30 a.m. the patient, while straining, became paralysed in the left side without any premonitory symptoms. The onset of the paralysis was sudden, without loss of consciousness, or even clouding of the intellect. Since then retention of urine. The paralysis of the leg passed off the next day. Entered March 3, 1907.

March 3.—Apoplectic appearance, face injected, slight cyanosis. Pulse regular, blood-pressure (Riva-Rocci-Landergren) 170. No enlargement of the heart towards the right; to the left it reaches the nipple line. The apex-beat not palpable. No murmur at the apex, but the aortic second sound, which is much accentuated and tympanitic in quality, is followed by a blowing murmur. No albumin or reducing substance in the urine.

The patient quite clear, but rather agitated. The speech hurried at times, somewhat blurred, but quite intelligible. Does not mix up words and has no difficulty in finding them.

Visual fields (rough examination) normal. No strabismus, pupils unequal, the left small, the right of medium size; they react very slowly to light and accommodation. Ptosis of the left eye. No sensory disturbances in the area supplied by trigeminus. Slight paresis of the left lower facial nerve. No deviation of the tongue. The left shoulder-joint can be moved a little, otherwise complete paralysis of the arm. No evident paresis of the leg. Reflexes of the arm absent, knee-jerk weak.

No impairment of the senses of touch or pain; localization of touch good. The patient perceives and describes correctly even minute movements of the paretic arm. On both feet slight uncertainty of the small movements of the toes.

The motor disturbance diminished rapidly in the two proximal joints, but it still persisted in the hand and fingers, when the patient was discharged. There was, however, at that time no complete paralysis. "Gesammtbewegungen" being performed, though with greatly diminished power. Reflexes still absent on the arm. No signs of sensory disturbances (thermal sensibility not examined). Condition of pupils unaltered. Discharged May 23.

The sudden onset of the apoplexy during straining, and the general state of the patient (face injected, cyanosis and hypertonia) indicate that a hæmorrhage might have been its cause. The rapid and complete restitution of the motility of the leg and a permanent paralysis of the arm shows that the lesion cannot have been capsular; the seat of the injury must have been in the cortex or in its neighbourhood.

In this patient syphilis had been insufficiently treated. The heart-symptoms of the patient indicated a disease of the aorta, perhaps an aneurysm, and it is very probable that the vascular lesion in the brain was due to the same cause. His pupils as well as his urinary troubles point to a metasyphilitic condition. But in this case there was no disturbance of the sense of passive movement or of cutaneous sensibility, in spite of a complete monoplegia in the arm; the localization of touch, too, was quite accurate. This case, therefore, corresponds to the preceding one, and supports the possibility of a complete paralysis without simultaneous disturbance of sensibility. Further, it must be noticed that at his discharge from the hospital the patient showed some power of "Gesammtbewegungen," though he was quite unable to execute isolated movements of the fingers. These facts indicate that these symptoms of cortical ataxia may appear without disturbances of sensation.

The reflexes of the paralysed side were in this case not even so brisk as on the sound side, but the metasyphilitic condition that I have assumed may have had some influence upon the reflexes.

CASE IX.—A. S. A., aged 72, married woman.

In the beginning of February, 1906, an apoplectic fit, dimness of vision towards the left; fell down. After a little while she was able to get up and walk, and the next day she could stand on her leg and move the arm. Was up and about within a few days; after that a rapidly increasing paralysis of the left side, apart from a new attack. This paralysis has gradually diminished in the arm.

January, 1907.—Mental condition good. Cranial nerves normal. Slight paresis of the left arm, most marked distally. Dynamometer left 17, right 31. Paresis of the leg considerably greater, still, she is able to perform all movements through nearly the same excursion as on the right side. The ordinary tests for ataxia reveal some uncertainty on both sides, but no appreciable difference between the two hands. Some awkwardness in finer movements of the left hand. Volitional movements are on the whole carried out well, but, especially when the patient is tired, with an irregularity resembling an intention tremor; this is more marked on the left side. No signs of ataxia (knee-heel test) nor of intention tremors of the movements of the legs.

Sensation of touch and pain normal in the arms, but on both legs some uncertainty to wool-touch. Perception of passive movements normal in the arms. In the toes slight uncertainty of the small movements on both sides. Perception of position good. With graduated disc, right hand against left.—

In the hip-plane an error of 5—6 cm. proximally and 2—3 cm. upwards.

In the xiphoid-plane an error of 6—8 cm. proximally, and 2—3 cm. upwards.

Left hand against right: a proximal error of 4—5 cm. in the hip-plane, and about the same in the xiphoid-plane.

The estimation is, however, difficult on account of the tremor; the error most likely to result from this cause is over-estimated in the latter observations.

The stereognostic sense intact.

Reflexes of the leg rather brisk; those of the arm considerably exaggerated. Slight plantar contracture.

On June 6, 1907, dynamometer right 35, left 18. Result of examination otherwise the same. The algesimeter of Alrutz showed no reduction of painful sensibility.

The seat of the lesion in this case was fairly certain. The leg was much more affected than the arm; and this fact argues to some extent against a capsular lesion—as well as against any central injury at all. But, on the other hand, the greater affection of the leg is the natural result of a lesion which has chiefly affected the cortical centre of the leg, and this supposition explains the symptoms. The view that the injury was a cortical one corresponds very well with the nature of the lesion, which, considering its slow appearance, was probably a thrombosis.

In this case the paresis was a brachio-crural one, and was most pronounced in the leg. In the two arms there was an intentional tremor, but this was more pronounced in the paretic side. In addition, orientation in space of the paretic arm was not so good as on the other side, a fact that I ascertained by an examination with the Horsley-Slinger method. Though there was some difficulty in making these observations owing to tremor, I could ascertain that the orientation in space of the arm was better with movement than at rest.

No disturbances of the sense of space were observed by the usual methods either in the leg or in the arm.

In the absence of disturbances of sensibility, despite disturbances of motility, this case corresponds very well to preceding ones; but it is not as satisfactory as these, as there may have been some disturbance of sensibility at first which disappeared, as a rather long time had elapsed after the stroke. The disturbance of orientation in space, despite the fact that the sense of position and of movement, as well as stereognosis, were intact, was remarkable, and in this respect this case diverges from that which follows it.

The reflexes in this case were exaggerated, as is usual with cerebral lesions, but they were greater in the arm, which was the less paralysed. As in a diffuse cortical or subcortical lesion, the leg recovers its motility, as a rule, better than the arm—the centre of the arm could not have been so much affected as that of the leg in this case. Disease of the artery that supplies the centre for the leg—*i.e.*, arteria cerebri anterior, which in this case was without doubt the seat of the injury—affected the centre of the arm only indirectly. I assume, therefore, that there existed a direct lesion of the leg centre, but only an indirect one in the arm centre.

In this case, too, I found a slight plantar flexion contracture. As the patient was not, however, properly treated for some time after the lesion, this contracture was very likely a passive one from the beginning, as there existed no contracture of the femoral muscles.

In this case an intention tremor was also observed, which appeared after the apoplectic fit, and may be regarded as a post-hemiplegic intention tremor (Infeld).

CASE X.—A. S. G., aged 45, female.

Migraine since youth, and for the last eight years rheumatic pains in the shoulders; numbness and stiffness of the hands, especially the left, for the same period. Otherwise well till December 10, when, after an attack of temporary weakness of the whole body, she felt the left arm gradually becoming paralytic. Was able to walk home without help.

Admitted December 13, 1907. Enlargement of the heart to the left, apex-beat strong; second aortic-sound accentuated; blood-pressure somewhat increased (Riva-Rocci-Landergren), 140. Urine contains traces of albumin and tube-casts, sp. gr. 1023.

Paralysis of the left arm; slight, but evident, paresis of the leg. No distinct paresis of the face, but the whole left side is somewhat smooth; ptosis on the left side. Appreciation of touch (wool) uncertain in the toes of both sides; otherwise normal. Painful sensibility (algesimeter of Alrutz), also defective in the toes, but elsewhere normal, and equal on the two sides. Localization exact. Sense of place (Weber's compass) unaffected.

Correct perception of passive movements in the toes, though a certain degree of uncertainty in very small movements.

Patellar and triceps-reflexes absent on the left side. Babinski's sign on the left

January 10.—Blood-pressure still higher, 180. Urine, no albumin, sp. gr. 1026. No paresis of the leg, face unaffected. Complete paralysis at the finger-, wrist-, and elbow-joints; very little movement at the shoulder-joint. No contractures; muscles of the left arm flaccid. Tendon reflexes slightly increased on the left arm and leg; Babinski's sign absent.

No alteration of sensation to touch and pain, except slight diminution in the toes; the same applies to the perception of passive movements. Examination of the orientation in space of the left hand with a graduated disc which, owing to the paresis, could only be done at rest, revealed a somewhat greater error than normally :—

In the hip-plane 0 cm. in distal, 1 cm. in cephalic direction.

In the xiphoid plane 3 cm. in distal, 2 cm. in cephalic direction.

In the shoulder-joint plane 0 cm. in distal, 5 cm. in cephalic direction.

On January 26.—Motor, tactile, and painful sensations, as well as perception of passive movements as before.

I must admit that the local diagnosis of this case is not certain, as I found paresis in the arm as well as in the leg; but, as the motility of the leg improved rapidly while the arm remained paretic, there was finally an isolated brachial monoplegia. Consequently the lesion was most probably a cortical one.

In this case the motor functions only were affected; the sense of touch and of pain, as well as the faculty of localizing, remained intact in the upper extremity; at least, there was no difference to be detected between the sound side and the paretic one. The very slight disturbances which were found on the toes and which did not alter during her stay in hospital, and the disturbance of the sense of passive movement, were, I suppose, only indirectly due to the cerebral lesion.

Orientation in space, as tested by the graduated plate, was found normal at the iliac level, but at the levels of the xiphoid and of the shoulder-joint the errors were slightly greater than normal.

CASE XI —C. H., aged 72, widow.

Typhoid fever at 30. For about eight years incessant tingling in the head and sometimes attacks of dizziness (had a sensation that the room was turning upside down) with difficulty in standing and walking. She usually lay in bed for a couple of days in these attacks; she vomited, but had no headache. Has never had a stroke before.

January 2, 1909. In the morning, patient complained of heaviness and numbness in the right arm; at midday, as she was busy cooking, she found that her left arm was weak; the weakness increased gradually so that she could not continue her work. No weakness in the leg (could ascend a staircase), but after a while difficulty in speaking. No giddiness or dimness during the attack; has been laid up since then. The power of the arm has gradually recovered, and the speech is completely restored. For about one week swelling and soreness, first in the left thigh, then in the right leg. Admitted January 25.

January 25—Pulse 100, systolic arterial pressure 160. Heart normal, trace of albumin. Thrombosis in the right leg.

Paresis of right face and right hand. Babinski on the right side. Senses of touch, pain, and of position and movement quite normal.

January 30.—Paresis of lower face conspicuous on moving, but little marked at rest. The tongue deviates a little to the right; other cranial nerves unaffected. The power in the shoulder-joint appears somewhat diminished in abduction and in elevation forwards; no diminution in other movements. In the elbow-joint the power is reduced in flexion and, though to somewhat smaller degree, in extension, but the movements are performed to their full range. The strength of all movements at the wrist is weak, especially supination and pronation. The power of each finger equally reduced (dynamometer right 2, left 3—10), but range of movement normal. Isolated finger movements can be made, but are slower in the right than in the left hand. The motility of the leg could not be tested on account of the thrombosis; at any rate, the patient has, on several occasions, been seen to move and lift up her right leg with so great facility that paresis may be excluded.

Sense of touch and of pain on both sides shows no reduction. Power of localization normal. Perceives exactly the direction of minimal movements in all joints of the upper right limb.

Stereognosis normal.

Orientation in space, right hand recording:—

Xiphoid level ...	1 cm. proximal ..	1 cm. caudal error
	½ ,, ,, ...	1 ,, ,, ,,
	1 ,, ,, ...	1 ,, kephal
Iliac level	0 ,, ,, ...	1 ,, ,,
	1½ ,, ,, .	1 ,, ,,
	1 ,, distal ..	0 ,, ,,

Left hand recording:—

Xiphoid level ...	0 cm. proximal ...	6 cm. kephal error
	2 ,, distal ...	7 ,, ,, ,,
	1 ,, ,, ...	6 ,, ,, ,,
Iliac level	2 ,, ,, ...	8 ,, ,, ,,
	1 ,, ,, ...	7·5 ,, ,, ,,
	1 ,, ,, ...	6·5 ,, ,, ,,

Tendon reflexes on the arms rather brisk, equal on both sides. Abdominal reflex absent on right side, feeble on left. Plantar reflexes of normal type on both sides.

February 2.—Motility and reflexes as before. Muscular sense undiminished. Senses of touch and pain somewhat uncertain in the distal parts of both hands (hands at the time cold).

Orientation in space examined by Horsley's method gave approximately the same results as in the previous observations, though the variation was now less.

Left hand recording:—

Xiphoid level 47/10, iliac level 46/10 cm. error

Right hand recording:—

Xiphoid level 14/10, iliac level 17/10 cm. error

Blix's method of testing orientation of active movements showed only slightly greater mistake on the paretic side:—

Right hand, eyes closed 4·5, eyes open 1·5 cm.
Left hand, „ 3·6 „ 1·2 cm.

February 11.—Motility as before, likewise reflexes. No disorders in the senses of touch, pain, nor in the muscular sense.

Orientation in space (Horsley-Slinger's method):—

Left hand recording:—

Iliac level 3·2 cm., Xiphoid level 3·2 cm. error

Right hand recording —

Iliac level 2·4½ cm., Xiphoid level 2·6 cm. error

Orientation of active movements (Blix's method): right hand, 1·3 cm.; left hand, 1·5 cm. error (eyes closed).

March 13.—Some paresis left in the hand. Horsley-Slinger's and Blix's methods give the same result on both sides. Reflexes as before.

April 11.—No definite paresis. Reflexes as before. No contracture.

Discharged April 16.

In this case there was evidently cerebral arterio-sclerosis; the slow, gradual onset of the lesion without affection of consciousness makes it probable that it was a thrombosis. The most important question as regards the localization of the lesion is, Was it in the cortex or in the internal capsule?

In both cases the arm may be the most affected, as was here the case. But the comparatively moderate paresis in the arm, and the absence of paresis in the leg, on the patient's falling ill, and, as far as could be judged by her spontaneous movements, three to four weeks later, while there was an obvious paresis of the face and tongue, puts a capsular lesion out of question. These facts point, on the other hand, to a cortical lesion involving, but not completely destroying, the lower and middle portions of the motor zone; the simultaneous affection of speech suggests an affection of Broca's gyrus.

It is interesting that a lesion which affected so extensive a region as the motor centres of the tongue, face, hand, and elbow, and, though to a less extent, that of the shoulder, left the muscular sense and stereognosis and symbolia quite unaffected.

On the other hand, the orientation in space of the paretic hand at rest was, at first, much worse than that of the sound one, whereas its orientation in movement—no matter if it was guided by the perceptions of position of the other side, by the sense of sight, or by visual memory—was normal, or almost so. Later, the orientation in space improved even in repose, and became as accurate as in the sound hand.

The tendon reflexes were brisk but equal on both sides, and no signs of contracture appeared during three months. Babinski's sign was not observed, but at the beginning there was some diminution of the abdominal reflex of the monoplegic side.

CASE XII.—C., aged 53, a farmer.

Four years ago left-sided hemiplegia with sudden onset without premonitory symptoms, which disappeared completely after a few weeks.

From the end of June, 1908, indisposed, with a tendency to giddiness and a feeling of weakness; has been feeling tired, difficulty in collecting his thoughts. On July 16, after violent exertion, severe giddiness for a minute or so. On July 18 the patient felt ill, was irritable and restless. Immediately after strong exertion sudden severe headache and giddiness. To prevent himself falling was obliged to lie down. Difficulty in speaking. Consciousness not quite lost.

Examination four hours later: Intellect clear; difficulty of finding words; can read vertical print. Paresis of the right side of the tongue and the lower part of the right face. Weakness of the right arm, and especially of the hand; but he is able to perform isolated finger movements. Motility otherwise unaffected.

No diminution of sensibility to touch and pain, or of the sense of position and movements. Stereognosis and symbolia normal.

Reflexes of the right arm absent; knee-jerk somewhat brisker on the right side: Babinski's sign on the right foot; abdominal reflex absent on the right side.

Heart somewhat enlarged—reaches to the left ½ cm. outside the nipple line. Accentuation of the aortic second sound. Pulse of high tension, regular, 52 per minute. Urine about 1,100 cm. in the twenty-four hours, specific gravity 1012, solitary hyaline and granular casts.

July 19.—Speech better. Facial paresis gone. Paresis of the arm and reflexes as on the 18th

July 20, at 1 p.m.—Speech normal. Paresis of the arm as before. *Reflexes of the right leg absent; no Babinski's sign, no abdominal reflex on the right side.* Seems inattentive.

July 20, at 8 p.m.—Wandering; paresis of the arm considerably increased; marked facial paresis. Speech slurred and unintelligible. Reflexes as at the last examination. Pulse 50, high tension, irregular.

July 21.—Intellect clear, speech difficult to understand, can read vertical print. Very marked paresis of the arm. Sensibility normal. Stereognostic perception unaffected. Reflexes of the arm as on the previous day. Babinski's sign on the right foot. Knee-jerk exaggerated on the right side.

July 23.—Paresis less marked; reflexes as before.

July 27.—Facial paresis almost gone. Paresis of the arm still evident. Talks without any difficulty.

August 6 and 19.—Speech normal. Handwriting as before the illness; still a trace of paresis of the hand; motility otherwise normal. Tendon reflexes somewhat livelier on the right leg; no Babinski's sign, abdominal reflexes normal. Reflexes of the right arm absent, of the left normal.

This man, aged 53, who had a stroke four years ago, was affected by another apoplexy after a period of rather vague general symptoms. It is very likely that the affection of his kidney—which must be considered as an interstitial nephritis on account of the small percentage of albumin, the few tube-casts and the low specific gravity of the urine—was a contributive cause of these apoplectic attacks.

The specific gravity of the urine, its reduced quantity, and the rapid disappearance of the cerebral symptoms would be reasons for assuming a purely uræmic condition. But this is not very likely, considering the limited monoplegic "Ausfallserscheinungen," absence of symptoms of irritation, and the preservation of consciousness. Moreover, the absence of the reflexes immediately after the attack strongly supports the assumption that the lesion was a vascular one, and its onset during exertion points to the same hypothesis. There is no doubt that in this case it was a hæmorrhage. Evidently the patient had several attacks, and the last two affected the same place.

As the paresis affected the arm, the face and the tongue, but not the leg, the diagnosis of a capsular lesion must be rejected, and the lesion must be localized in the motor cortex or in its neighbourhood.

The slight symptoms of aphasia might argue in favour of a localization of the injury in the neighbourhood of the speech centre, but the affection of speech was slight and temporary. It is possible that the injury affected the frontal lobe; the motor zone was involved only to a slight extent. This hypothesis corresponds well with the rapid recovery of the disturbances of motility.

Even four hours after the attack sensibility was unaffected, at least as far as touch, pain, position and movement were concerned. Stereognosis and symbolia were also intact.

The condition of the reflexes during the course of the disease was interesting. At first the reflexes of the arm were absent, but the patellar was exaggerated and the plantar reflex gave an extensor response, due to the first slight lesion. Later I observed an absence of the reflexes in the leg; this proved to be the initial symptoms of a new lesion. Two weeks after the second stroke the patellar reflex was again obtained, and then it was stronger than normally. On the contrary, a month after the attack, during the stage of residual paralysis, the reflexes of the right arm had not yet reappeared.

(*b*) *Sensory-motor Symptoms* (*Cases XIII. to XVIII.*).

CASE XIII.—E. L., aged 66. Housewife.

Previous health.—On March 13 at noon the patient became giddy and weak in the right arm, so that she had to leave off her work. She attempted to fetch help, but after a few steps the leg gave way and she fell. She noticed that her speech was blurred, and that it was difficult to pronounce even isolated words. Consciousness was retained throughout.

On admission, March 14, 1907, the following notes were made:—

No enlargement of the heart to the right; to the left the dulness reaches to ½ cm. inside the nipple line. Accentuation of the second aortic sound. Peripheral arteries somewhat rigid. Blood-pressure (according to Riva-Rocci-Landergren) 180, urine contains trace of albumin. No hemianopsia. Movements of the tongue unaffected. Slight paresis of the lower face. Considerable paresis of the right arm. Babinski's sign present. No abdominal reflex on the right side. Tendon reflexes somewhat increased on the right side. Muscular sense dulled.

March 16.—Intellect quite clear. No aphasia nor dysarthria. No hemianopsia. Movements of the eyes unaffected; slight paresis of the left lower face; no deviation of the tongue.

Severe paresis of the right arm, only small movements of the finger-, wrist-, and elbow-joints being possible. The power of the leg somewhat reduced, especially at the hip- and knee-joints, but much less so than in the arm.

Tendon reflexes of the right arm and leg slightly increased, Babinski's sign on the right foot, no abdominal reflex on the right side.

Cutaneous sensibility of the face normal. On the *right* arm a cotton-wool-touch is not appreciated as high as a little below the shoulder; a finger-touch to about the elbow. On the *right* leg finger- and wool-touch are felt at the same level—viz., at the knee. In the *left* hand and arm no impairment of tactile sensation. Perception of cotton-wool-touch is in the lower third of the left calf and in the foot uncertain; a light finger-touch on the other is recognized here. On the *right* hand no discrimination between head and point of a pin. On the distal parts of the feet (about equal on the *right* and *left* side) considerable uncertainty. The sense of deep pressure seems everywhere well retained, but is less on the *right* hand. The patient perceives the vibrations of the tuning-fork everywhere, but they seem feebler on the *right* arm. No difference in the legs.

Passive movements of about 90° are correctly estimated at the *right* shoulder-joint, but great uncertainty in movements of 45°. No appreciation of passive movements of the elbow-, wrist-, and finger-joints. Movements of the knee-joint of about 30° or more are correctly described, but the appreciation of even the largest movements of the foot and toes is very uncertain.

On the *left* side the patient describes wrongly or vaguely even large movements—of about 15°—of the finger- and wrist-joints. Movements of the shoulder- and elbow-joints are correctly described. There is almost the same degree of uncertainty of the movements of the joints of the foot and toes on the *left* side as on the *right* one.

The appreciation of position of the *right* arm as well as of the leg is absent; the patient is able to find the paretic hand with the non-paretic one, only by groping; he is unable to imitate the postures of the right arm or leg.

March 20.—Motility of the shoulder- and elbow-joints somewhat improved, the patient being able to raise the whole arm from the supporting surface. Paresis of the hand about the same. The power of the leg fairly good.

Cutaneous sensibility: Appreciates a touch with cotton-wool at about the level of the elbow, a finger-touch over the whole of the fore-arm and on the dorsum of the hand, but not on the fingers nor on the palmar aspect of the wrist.

On the lower extremity a cotton-wool-touch is appreciated below the knee and on the dorsum of the foot, and a finger-touch on the big toe. The patient is able to differentiate the head from the point of a pin except on the distal phalanges of the second and third, on the whole of the fourth and fifth fingers, and on the ulnar border of the hand. The algesimeter of Thunberg is not appreciated at 3 grm. till just below the elbow-joint. When the liminal value is passed, the sensations produced by the same weight are said to be stronger on the right side.

Sense of deep pressure as before.

Passive movements of about 45° are correctly perceived at the shoulder-joint. The other joints of the right upper-extremity show the same condition as before. She has exact perception of minimal movements of the knee-joint, but at the ankle-joint there is uncertainty of even maximal movements. Perception of position of the *right* upper extremity still totally absent, that of the leg fair.

On the *left* side some uncertainty of passive movements of the toes. Otherwise *no* disturbance of sensibility noticeable on the left side.

April 15.—Paresis of the shoulder- and elbow-joints now considerably less. Dynamometer on the right side 2, on the left 25. The power of the leg is not much weaker on the right side than on the left. Reflexes as before, no contracture. The patient appreciates cotton-wool everywhere except on the dorsal surface of the four ulnar fingers and on the palmar surface of the ulnar half of the wrist and four ulnar fingers. Finger-touch is, however, felt here. On the same parts she is unable to differentiate between the head and point of a pin when lightly touched. Does not appreciate Thunberg's algesimeter with a weight of 10 grm. on the distal phalanges and not until the weight has reached 6 grm. on the palmar aspect of the ulnar half of the metacarpus. Sense of pain otherwise not impaired.

On the toes of both sides a touch with cotton-wool is not felt.

Power of localizing a touch on the fingers considerably impaired; as a rule the touch is localized towards the metacarpo-phalangeal joints, but in the little finger sometimes even higher. (The result of the test was the same whether the patient had to point at the place touched or had to describe it.)

Tested with Weber's compasses, the patient discriminated the points at a distance of 10-12 mm. only on the distal phalanges of the right side, at 3-4 mm. on those of the left.

Appreciation of passive movements still almost absent in the joints of the hands and fingers. Appreciates correctly movements of about 20° at the elbow-joint and minimal ones at the shoulder-joint. Even the smallest movements of the knee are exactly described. Movements of the ankle-joint, under about 30°, are not perceived with any degree of certainty; the uncertainty is still greater in the joints of the toes. In the left foot there is some uncertainty in smaller movements of the toes.

Orientation of the position of the arm still very bad. Examination with Horsley's disc impossible.

The recognition of the nature of objects by touch and the power of identifying them lost.

May 18.—Motility of the right arm considerably increased, especially proximally. Dynamometer: right 5, left 26. No paresis of the leg noticeable.

Reflexes still somewhat increased in the right arm and leg. Babinski's sign on the right foot. No abdominal reflex on the right side. No rigidity or contracture.

Cutaneous sensibility as at the last examination. Localization is, however, now better. No impairment, at present, to be made out on the palm of the hand. A touch on the dorsal aspect of the distal phalanges is located on the second interphalangeal joint, or somewhat more proximally on the middle phalanx On the whole of the little finger and ulnar border of the hand proximal reference of 2—3 cm.

No improvement of the perception of passive movements of the arm or hand, but possibly a slight improvement in the joints of the foot and toes.

Stereognostic perception as before.

Discharged May 19. June 12, condition the same.

That the lesion in this case was a vascular one is evident. The gradual onset of paresis in the arm and then in the leg, as well as the maintenance of consciousness, are the symptoms which generally indicate a thrombotic softening. The peripheral arterio-sclerosis also points to a thrombosis.

The existence of a monoplegia is decidedly in favour of a lesion in a region where the different centres or their efferent fibres are distant from each other. It cannot therefore have involved the internal capsule or its neighbourhood, but probably lay in or near the cortex; thrombosis is also most common in the cortex.

The initial considerable diminution of cutaneous sensibility and of the sense of position and movement is interesting. In this case it occurred even on the non-paretic side, and was most pronounced in the leg. The sense of position and of movement of the hand was also affected. This symptom was not owing to a lack of attention on the part of the patient, but must have been due to her lesion. Considering its sudden disappearance, it must be either attributed to a diaschisis or looked upon as a result of the bilateral innervation of these functions.

Fr. Müller has already observed a diminution of the cutaneous sensibility in the lower extremity of the sound side; he attributed it to a bilateral representation. But a bilateral innervation of the sense of position and of movement of the arm and hand does not agree with the well-known fact that these functions usually remain the most severely affected of all sensory functions. On the contrary, a distant affection might very easily occur at the onset of a vascular lesion of the brain; and thus it is possible that a function as complicated as the sense of position and movement might be disturbed.

The similarity of these symptoms, which in my opinion were functional, to the symptoms produced by an organic lesion is very interesting. I have noted the same condition in Cases XVII. and XXI.

The affection of the monoplegic arm consisted in paresis and diminution of the sense of position and movement. The latter were the most intense and persistent; for instance, when the power of movement at the elbow- and shoulder-joints had almost completely recovered, the patient was able to appreciate only large movements at these joints. The faculty of localization, too, which usually runs parallel with the muscular sense, had almost recovered when the muscular sense was still greatly disturbed.

There was also a persistent partial distal anæsthesia—*i.e.*, an anæsthesia of ulnar type—during a period the case was under observation.

In this case, too, there was a certain degree of "hyperalgesia." The liminal intensity was much higher on the paralysed than on the sound side; but when the liminal intensity was passed the sensations became more painful in the paralysed side than in the sound one, and these symptoms appeared even at an equal weight on the two sides.

The reflexes were somewhat increased in the paralysed side, and remained so during the whole time of observation; even three months after the onset there were no signs of contracture.

CASE XIV.—K. G. N., aged 36, labourer.

Was an in-patient April 28 to September 29, 1903, for cerebral syphilis and pulmonary tuberculosis. Ill since the summer 1902, when he had an apoplectic stroke while under treatment at a sanatorium. Dulness and prolonged expiration over both apices behind. No albumin.

Aphasic, paralysed in the right arm, paretic in the right leg; deviation of the tongue towards the left. Pupillary reaction absent, the pupils being widely dilated.

In January, 1907, his condition was as follows. No hemianopsia. Slight paresis of the lower part of the right side of the face. Deviation of the tongue towards the left; the left half considerably atrophied. Right trapezius weaker, and this muscle, as well as the deltoid, is wasted. Is unable to raise the right arm fully, but offers fairly good resistance, although weaker than on the left side

Flexion and extension of the elbow-joint performed slowly and without precision, as if he had to work against a considerable resistance; the movements are better performed when controlled by vision. The movements are full in range and fairly strong, but they are weaker than those of the left limb; no atrophy, the muscles of the forearm being well developed. The excursion of the movements of the wrist normal.

The hand is usually held lightly clenched, the thumb within the finger. Active movements of all the finger-joints can be performed to the extent of about half a right angle, but only "Gesammtbewegungen"; passive movements unrestricted. Dynamometer: right hand 4, left 50.

Power of the right knee good; no definite difference when compared with the left leg. The foot is held in moderate plantar flexion. Dorsal flexion can be performed with fair strength; movements of the toes a little weaker on this side. The gait of the right leg is spastic.

Feels a cotton-wool touch everywhere except on the distal phalanges of the right hand, the outer one-third of the right foot, and over the distal phalanges of the right as well as of the left foot. No impairment of the sense of pain.

At the shoulder-joint movements of about 5° can be passively performed without the patient being able to estimate their direction with certainty; on the other hand, he indicates when a movement takes place, even when this is minimal. The elbow-joint may be moved without the patient appreciating the movement. At the joints of the wrist and fingers no exact appreciation of the direction even of extreme movements, on the other hand, he indicates accurately as soon as a movement takes place, even small movements.

At the right hip and knee all movements are at once appreciated. The muscular sense of the ankle-joint obviously less good, the patient being uncertain of the direction of movements up to 30° to 45°. The same applies to the toes. On the left side no impairment of the perception of passive movements was observed

Imitation of passive movements of the paretic side with the sound arm is very inaccurate, but it is better in the leg. If, on the other hand, the patient is asked to imitate with the paretic side passive positions of the sound side, he does so accurately on the leg, but makes gross errors in the arm.

The right hand is clumsy, slow, and uncertain in its movements, but the error in attempting to bring the forefinger to the nose or to the opposite forefinger is usually not greater than 5 to 10 cm.; there is no regularity in the direction of the error. With left hand against the right there is constant proximal error of a couple of centimetres in the finger-to-finger test; he points, as a rule, towards the centre of the forearm or often higher. The finger-nose test, on the other hand, is performed accurately with the left hand.

The knee-heel test performed somewhat less well with the right leg than with the left. This is evidently due, partly at least, to the spasm.

Considerable disorientation of the right arm, especially of its more distal parts; orientation of the right leg good.

Localization of touch in the upper arm good; in the forearm and hand it becomes less and less good distalwards, localizes a touch on the dorsum of the hand and fingers most often to the wrist, seldom more proximally. In the leg no difference worth mentioning compared with the left.

The distances at which the two points of the compasses can be discriminated are shown in the following table.

A = dorsal surface; B = volar surface.—The values of the left side are enclosed in brackets.

		Phalanx.								Phalanx.					
A. Finger	1	I	15	(12)	II	13	(6)	B.	1	I	9	(7)	II	9	(2)
,,	2	,,	14	(15)	II	19	(8)	,,	2	,,	8	(9)	II	7	(4)
					III	15	(4)						III	5	(2)
,,	3	,,	—	(15)	II	15	(9)	,,	3	,,	9	(9)	II	8	(4)
					III	15	(4)						III	15	(1)
,,	4	,,	17	(6)	II	12	(8)	,,	4	,,	8	(10)	II	8	(5)
					III	11	(4)						III	12	(2)
,,	5	,,	—	(14)	II	10	(10)	,,	5	,,	8	(7)	II	9	(5)
					III	13	(4)						III	12	(2)

Metacarpus	1	24	(24)	9	(9)
	2	23	(22)	9	(11)
	3	28	(25)	10	(11)
	4	25	(21)	11	(10)
	5	26	(25)	9	(10)

Carpus, right 29, left 22.

Tendon reflexes somewhat increased in the right arm and leg. Babinski's sign present on the right side. Pupils unaltered. Intelligence considerably blunted. Motor aphasia.

A man, aged 32, was affected by an apoplexy, producing a right-sided hemiplegia and motor aphasia. That the underlying cause was syphilis is probable, considering the age of the patient as well as the condition of the pupils at an earlier observation. The patient, who was specifically treated after the stroke, has shown no new symptoms other than those associated with his hemiplegia, and no new attack has occurred.

On account of the persistence of pure motor aphasia, the attack was probably cortical. This diagnosis is rendered more probable by the existence of facial paresis, the approximately complete paralysis of the fingers—not only loss of isolated movements—and the very pronounced paresis of the wrist-joint, though there still remained a fair power at the elbow-joint and the absence of diminution of the gross power of the leg.

The paresis of the left half of the tongue and its atrophy show that there must have been another lesion which involved the hypoglossal nerve or its nucleus. This may be a tabetic symptom.

His hypæsthesia in the lateral border of the foot—which was not combined with analgesia, but was associated with a diminution of the muscular sense of the ankle-joint—may point to another lesion too. But, considering the type of sensory disturbance, this lesion cannot have been a spinal one, and corresponds very well to the symptoms that result from a cortical lesion. It seems to me to be very probable that these sensory disturbances were due to the lesion that produced the hemiplegia and the aphasia.

The lesion that was the cause of his paresis, judging from the "Ausfallserscheinungen," affected a motor speech-centre as well as the centres of the arm, or at least that of the hand.

I may draw attention to the fact that in the arm the disturbances of motility and those of muscular sense were almost equally pronounced in the respective joints. Both were moderately disturbed in the shoulder- and elbow-joints, but more profoundly in the wrist- and finger-joints. The disturbance of muscular sense, however, was more complete in the latter than the disturbance of motility, and in the ankle-joint there was a diminution of the muscular sense without any appreciable amount of paresis. Thus the disturbance of the muscular sense was in this case almost parallel to that of motility, though somewhat more intense.

The orientation in space of the paretic arm on active movement was good in comparison with its orientation at rest. Thus the error at the finger experiment, executed with the right hand recording, was noted to be about 5 cm. to 10 cm.—an error which might be partly attributed to the paresis. When the experiment was performed in the other direction the error was very much greater. The error, however, appeared with a certain uniformity, inasmuch as the left hand was almost always lifted towards a point too far headward and proximal.

The orientation of the movements of the right hand, too, were not so bad, considering the evident disturbances even of the proximal joints, and this case confirms, in my opinion, Dejerine's [46] experience that the orientation of active movements is usually superior to that of the passive ones. Owing to the pronounced paresis it was not possible to decide if there was some cortical ataxia in the hand. The peculiar slowness of the movements of the elbow-joint might be, perhaps, an expression of a disturbance of this kind; but it may have been due to an intellectual defect.

It is interesting to note that the localization of tactile stimuli was reduced in the arm, though the disturbance of tactility was inconsiderable. The power of localization examined by Weber's compasses appeared to be much better than at an examination with successive stimulation. Whether this singular condition was due only to mental dulness, or if it has some special significance, I must leave to further discussion.

CASE XV.—A. P. L., aged 56, bootmaker.

Belongs to a family of drunkards. Himself a drinker since youth. In 1884 aching pains in the joints; later paræsthesiæ and a feeling of softness under his feet, as well as clumsiness in performing finer movements. In 1905 pneumonia; during convalescence tachycardia and dyspnœa. Has felt strangely listless and tired during last month. On November 28, 1906, when at work, the knife fell from his right hand, and it was only with control of the eyes that he was able to hold it for a short while. Paræsthesiæ of the arm and hand, as well as fine twitchings of the hand. Later on paresis of the arm as well as twitchings, but no paresis of the leg. He also noticed a shadow obscuring one-half of the visual field. Headache. Admitted December 1.

December 1.—No hemianopsia; slight paresis of the right side of the face. All movements of the shoulder- and elbow-joints can be performed to full excursion, but with only little strength, especially at the elbow-joint. Is to a certain degree able to move the wrist. Paralysis of the fingers. Slight paresis of the leg.

Tactile sensation diminished on the hand; does not appreciate a finger-touch on the dorsal aspect of the terminal phalanges; no appreciation of cotton-wool on any part of the fingers, nor on the thumb. On the palmar aspect a finger-touch is everywhere felt. On the terminal phalanges he cannot differentiate between the head and the point of a pin.

Exact perception of even minimal movements of the shoulder- and elbow-joints. At the wrist and joints of the thumb great uncertainty, but movements of 30° always correctly described. In the other fingers still greater diminution, increasing towards the ulnar side. No exact appreciation of movements of 45° in the four ulnar fingers.

At the hip- and knee-joints no uncertainty. At the joints of the foot and toes uncertainty of minor movements, but it is equal on both sides. Passive movements of the paretic hand are badly imitated by the non-paretic one. The finger-finger test against the paretic hand is performed badly, with an error of 20 cm. to 30 c.m., usually in a proximal direction, or towards the middle line. On the other hand, he places the left index-finger without any greater error on the tip of the nose, the nipple, &c. The knee-heel test is carried out with out precision on both sides.

No reflexes on the right arm; very weak on the right leg. No plantar or abdominal reflex on the right side.

December 4.—Headache, pains, and paræsthesiæ over the whole body. Slight paresis of the lower part of the face persists. Strength of the elbow much better, and he is also able to perform a number of minor movements with the fingers. No paresis of the leg.

Feels cotton-wool everywhere except on the terminal phalanges of the right hand, and here he cannot certainly appreciate the differences between the head and point of a pin.

Recognizes correctly minute passive movements of the shoulder-, elbow- and wrist-joints, but is uncertain of small movements of the joints of the thumb. In the other finger-joints the condition is unaltered.

Passive movements are well imitated, except those of the fingers.

Pain sensibility of the legs as before. Stereognostic power not much impaired; he discriminates the nature of objects and is able to identify them.

Reflexes normal on both sides.

December 14.—Motility, and the appreciation of passive movements of the hand somewhat improved.

December 22.—Strength of right hand increased, no weakness of the shoulder-joint, slight weakness of the elbow-joint. Dynamometer: right 14, left 50.

Tactile sensation as before. Appreciates minimal passive movements of the wrist and of the carpo-metacarpal and metacarpo-phalangeal joints of the thumb. A considerable degree of uncertainty of the movements of the other joints up to 30°. Stereognosis is unaffected.

On January 6 a fit; headache, restlessness, paleness, irregular action of the heart, convulsive movements of the left side. After the attack the paresis of the right arm was greater than before, but it diminished during the next few days. There was no increase of reflexes.

January 8.—Paresis considerably increased since December 4. He is uncertain of movements of about 15° at the wrist-joint, and of even large movements in all the joints of the fingers.

March.—Tactile and painful sensibility and the impairment of muscular sense as before, equally marked in all the finger-joints.

No increase of reflexes; no contracture.

Discharged March 21.

This was the case of a man, aged 56, who had two hemiplegic strokes, in each of which the lesion was cortical. The premonitory symptoms of irritation and the incomplete monoplegia favour this diagnosis. The paresis after each stroke was similar and affected the right arm. The disturbance of tactility consisted, as is usual in such cases, in slight distal hypæsthesia. (The power of localization was not examined in this case.) The condition of the muscular sense was more remarkable; after the first stroke it was more disturbed in the distal joints, but the sense of movement was evidently less disturbed in the joints of the thumb than in the other fingers. After the second attack the muscular sense showed a uniform and rather pronounced diminution in all the finger-joints.

Such a case points to the existence of a special centre for the muscular sense of the thumb, which can remain relatively intact when the motility both of the thumb and of the fingers is abolished and the muscular sense of the fingers severely affected. The second stroke, however, produced a disturbance of the muscular sense of the thumb.

It is interesting that stereognosis was intact, in spite of paresis and disturbances of muscle-sense. The reflexes, which were absent on the paralysed side after the first fit, were not increased after the second.

No contractures developed during an observation of three and a half months.

CASE XVI.—I. G., married, aged 56.

From the beginning of 1890 complained of tiredness and headache coming on at times, and sometimes followed by vomiting; swelling of the legs and on one occasion also of the face. Albuminuria was found in 1891.

Early in January, 1905, weakness of the left arm and leg. On January 7 some headache, chiefly on the left side. Left hand somewhat weaker than the right; according to a statement made later by the patient, there was at this time no disorder of sensibility when tested with a pin. Patellar-tendon reflex possibly a little brisker on the left side; Babinski's sign on the left; no ankle-clonus. Blood-pressure 165. Trace of albumin and granular tube-casts in urine.

Later the paresis passed off completely.

Early in January, 1907, a severe attack of giddiness and vomiting; after the attack paralysis of the left hand, but, according to her statement, no impairment of sensibility, not even immediately after the onset. On examination a few days later by Professor Petrén, there was paralysis of the left hand without any disturbances of sensibility to touch or pain or of muscular sense. Triceps reflex absent, patellar-tendon reflex weak on the same side.

The paralysis passed off completely during the next month.

At the end of May, 1908, another attack with giddiness, headache, and vomiting, return of the paralysis of the arm. This time, however, the sensibility of the hand was temporarily lost for a few days.

On July 20 I examined the patient: Power of movement at the shoulder-joint fairly well retained, rotation outwards only being much diminished; considerable weakness of extension of the elbow, less of flexion. Dorsal flexion of the hand very feeble, palmar flexion much better. The range of the movements of the thumb good, though all were greatly reduced in strength. Flexion of the three radial fingers fair, but the power of extension very poor, especially in the middle and ring fingers. The little finger is almost completely paralysed. No paresis of the leg.

A cotton-wool-touch is felt everywhere on the hand and fingers. The muscle sense is considerably affected in the little finger, less so in the next two, and is normal in the index-finger and thumb. The perception of flexion is somewhat impaired at the wrist-joint, but that of extension is good. No impairment of the muscle-sense at the elbow-joint. Localization on the hand uncertain; as a rule, the patient refers too far to the radial side, especially to the two ulnar fingers, but as often distalwards as proximalwards.

No contracture. Reflexes of the arm rather lively and those of the leg considerably increased.

In this case of interstitial nephritis repeated cerebral seizures occurred after a rather long period of symptoms suggestive of uræmia. All these attacks affected the left arm, and finally caused a pronounced and permanent paresis. I could obtain very little information about the first attack, but the account of the patient proved that the lesion had been very moderate, and that, at all events, the sense of pain was not much affected.

After the attack in November, 1907, the paresis was very pronounced at first, but some days later there were no disturbances in the sense of touch, pain, or of position or movement. The paresis almost disappeared.

Disturbances of cutaneous sensibility were not observed till the third stroke, and these remained for less than two months. The defect-phenomena, motor as well as sensory, were of the partial distal type, in both cases ulnar; but the affection of motility was a little more extensive than that of sensibility. Thus this case shows very clearly how repeated small vascular lesions affecting the same motor region gradually affect even the sense of position and movement. The uniformity between the disturbances of the sense of position and of movement and those of motility greatly favours the view put forward by Mills and Weisenburg, that the motor and sensory centres of the fingers are to be found at the same horizontal level.

The sense of position and movement at the wrist showed a particular condition—viz., a better appreciation of movements of extension than of flexion. I have noted a similar condition in Case I. It is remarkable that in this case (XVI.) the patient showed a diminution of sense of movements in the wrist, but not in the thumb or index-finger. It must be observed, however, that the patient used to keep her paretic hand hanging outside the sling by the aid of which she supported her arm; and this may have had some influence, inasmuch as the patient less easily perceived movements that were executed in the direction in which gravity usually acted than movements in the opposite direction.

The "simple" cutaneous sensibility remained quite intact in this case, but there was an error in localization in pre-axial directions on the ulnar side, *i.e.*, the power of localization was diminished in the same part as the sense of position and movement.

Finally, I must call attention to the loss of isolated movements which were observed in the index in spite of maintenance of the sense of position and movements.

The reflexes were absent a few days after the lesion, evidently a result of diaschisis; but two months after the second attack they were slightly exaggerated, more so in the non-paralysed leg than in the paretic arm.

CASE XVII.—E. F. J., aged 39, carpenter,

Measles in childhood, otherwise good health. On April 2 (eight days before admission) he remained in bed half the day on account of a cold, with coryza, headache, and tenderness all over the body, especially in the legs. The headache and nasal catarrh persisted, but he was well otherwise until April 6; he then felt weakness of the right hand, not preceded by twitching or paræsthesiæ, and had to lay down his tools. The weakness only lasted for a few minutes, but returned three times on that same day. During the next day he had a Jacksonian fit, beginning with convulsive movements of the forearm, which spread to the upper arm and the right half of the face and trunk; scarcely any movement of the hand. Within an hour two more attacks occurred of a similar character, followed by paresis of the arm and paræsthesiæ from the neighbourhood of the wrist to the right side of the body.

In the morning of April 10 he had a series of fits which lasted in all three hours and a quarter; the first began with twitching of the face, but all the others started in the forearm. After the attacks the arm was paralysed.

On admission into the hospital on the same day there was paralysis of the right arm and paresis of the lower part of the right side of the face. The strength and motility of the right leg were good.

At 6 p.m.—Mind quite clear, no headache, and no stiffness of the neck. The right half of the face somewhat weak. A marked degree of paresis of all the joints of the right arm, but nowhere complete paralysis. No paresis of the leg. No impairment of tactile sensibility or of localization; the patient appreciates correctly even minimal movements of all the joints. Reflexes normal.

April 11 at 12.30 a.m.—A fit (which I observed); the clonic movements were sharply confined to the shoulder- and elbow-joints, no twitching of the face or of the muscles of the hand. Paræsthesiæ of the arm during the attack. After the fit paralysis of the arm for about one hour. The perception of passive movements was considerably reduced at the wrist and elbow-joints immediately after the attack; not even maximal movements were appreciated with any degree of certainty; in the fingers the result was somewhat uncertain, but large movements were sometimes fairly well recognized. Appreciated cotton-wool-touch everywhere except on the most distal parts of the fingers. Half an hour later movements of the elbow-joint of 30° were correctly recognized and no affection of touch was present. After two hours no affection of sensation.

On April 11, 4.30 to 8 p.m.—Another attack (observed half an hour after its commencement); violent convulsive movements of the arm and forearm, especially of the flexors and pectoral muscles. Bilateral contraction of the sterno-mastoid and abdominal muscles. Twitchings of the muscles of the right thigh and slight clonic contractions of the facial muscles. The thumb was not involved and it could be moved voluntarily during the attack. After the fit the same impairment of tactile sensation as after the previous one. Total ignorance of the position of, and of passive movement in, the right arm. Uncertainty of small movements in the joints of the foot and toes, the sense of position in the leg otherwise fairly correct. No paresis of the leg, but the arm was paralysed.

April 12.—Motor paralysis of the right upper extremity except the thumb, all movements of which could be performed, although slowly and feebly; some paresis of the foot. Perception of passive movements of the wrist almost lost, and it was considerably reduced in the joints of the fingers and elbow, possibly, too, at the shoulder-joint. Small movements of the toes are badly perceived on the right side. Some uncertainty and subjective impairment of the tactility in the right hand and fingers.

April 13.—Motor aphasia. April 14.—Total hemiplegia; owing to the aphasia and some impairment of intellect a detailed examination of the sensibility is difficult. There were evidently, however, hypæsthesia and hypalgesia and a total loss of the sense of position of the right limbs. Later, symptoms of meningitis developed. Death occurred on April 18.

Post-mortem.—The whole left hemisphere appeared flattened and larger than the right, especially the central gyri, which measured 3 by 2 cm. on the left side compared with 1·2 by 1 cm. on the right. The central gyri and the adjacent portions of the frontal and parietal lobes were discoloured and soft; fluctuation was obtained in the posterior central gyrus and the anterior part of the parietal lobe.

On frontal section multiple softenings and liquefied areas of the size of a hazel-nut or larger, partly connected with each other and surrounded by necrotic tissue, were found in the white matter of the central convolutions and parietal lobe. The largest of these cavities, irregularly shaped and bigger than a walnut, was situated in the parietal lobe and gyrus centralis posterior, reaching the surface of the brain at the posterior border of the latter. The lesions extended downwards as far as the basal ganglia, but did not involve the internal capsule.

The following were the cerebral symptoms in this case. Attacks of temporary paresis in the right upper extremity, especially of the forearm. Fits of irregular type (beginning on one occasion in the face), which were followed by a permanent paresis in the arm. At first there was no disturbance of the sense of touch, of localization or of the sense of position and movement; but after a series of Jacksonian fits, temporary but considerable disturbance of the latter was found, with very little disturbance of the sense of touch.

Considering the very large extent of the lesion, this case only confirms the well-known fact that lesions in the centro-parietal region give sensory-motor symptoms. It was not possible, however, to make a detailed examination of the sensibility during the last days of life. The case also shows that a lesion elsewhere rapidly progressive may paralyse the arm without producing any disturbances of the sense of touch, of position, or of movement.

Post-convulsive sensory paralysis may be common, though it is not so frequently seen as post-convulsive motor palsy (Burr, Binswanger, Mann; see also my Case XXI.). I was, however, astonished to find in this case that the sense of position and of movement was chiefly affected, and touch but very slightly. This fact proves either that the localization of these functions must be different, or, if they have the same localization, that the sense of position and movement is more easily affected than the sense of touch. Unfortunately, I did not examine the patient's power of localizing touches.

I wish to call attention to the fact that the temporary post-convulsive disturbances of sensibility resembled closely the usual stable disturbances of sensibility. I think that this condition ought to be observed in typical Jacksonian epilepsies in order to study further the symptomatology of the cerebral disturbances of sensibility.

CASE XVIII.—J. V. K., aged 53, joiner.

Patient had previously enjoyed good health. Since the summer of 1907 he had shown signs of œsophageal stenosis, which developed slowly, and are now severe.

Without premonitory symptoms, patient suddenly became paralysed in his right hand in the beginning of October, 1908. One morning on waking he felt the fingers of this hand weak, but the weakness did not, as far as he can recollect, extend to the wrist, and certainly not to the elbow- and shoulder-joints. At the same time he had a feeling of numbness and paræsthesia in the whole hand except the thumb. The sense of touch, too, was reduced in the fingers, but gross painful and stimuli were always appreciated. His right hand was somewhat fumbling and awkward in handling even light objects (not only small ones); he was scarcely able to wield a hammer with it, and when he held a nail with his right hand and the hammer with the left the right hand was observed to be unsteady. He could, however, use his right hand in eating, and had not himself observed any awkwardness of it.

November 26.—Palmar and dorsal flexion, pronation and supination of the right hand are performed with considerably reduced power. Dynamometer, right side 0, left 25.

Sense of touch lost on the palmar surfaces of the fingers (cotton-wool); sense of pain diminished in the terminal phalanges; sense of position and movements uncertain in the metacarpo-phalangeal and interphalangeal joints. In the left hand no disorders of sensibility.

December 5.—No paresis in the wrist ascertainable. The movements, especially flexion of the thumb as well as of the other fingers, feebler than on the other side, but plainly better than on the date of the last examination.

December 10.—No paresis of the movements of the elbow- or shoulder-joint. In the wrist no paresis demonstrable in palmar flexion, pronation, or supination, but the dorsi-flexion is somewhat feebler on the right side. The flexion, extension, abduction, and adduction of the fingers much feebler on the right than on the left side, and the reduction in power seems to be equally marked in all those movements; the paresis in the fingers is far more pronounced than on December 5. The power of isolated movements in the fingers is normal.

No disorder of touch or localization could be ascertained. Muscular sense plainly uncertain in the little finger, elsewhere intact.

December 14: Dynamometer on the right side 14, on the left 28. The paresis in the fingers about as marked as on December 5. Touch and muscular sense normal. Stereognosis and symbolia, which are carefully examined, were intact. Orientation in space (Horsley's disc) normal.

Left hand recording :—

Xiphoid level error 1·8, oftenest in kephal direction.
Iliac ,, ,, 1·4, ,, ,, ,,

Right hand recording .—

Xiphoid level error 1·6, direction varying.
Iliac ,, ,, 1·4, ,, ,,

Active orientation according to Blix-Öhrvall's method :—

Right hand, eyes open error 0·4 cm.
,, ,, ,, shut ,, 3·3 ,,

Left hand, eyes open error 0·5 cm.
" " " shut " 3·0 "

December 15: Dynamometer left 35, right 20.

December 27.—No paresis in shoulder- or elbow-joints. Palmar flexion, pronation, and radial and ulnar flexion normal, but dorsal flexion and supination somewhat feebler on the right side. All finger movements feebler, especially extension and flexion. The latter cannot be made to full range; he cannot bring the finger-tips to the thenar and hypothenar eminences. Finer associated and isolated finger movements are normally performed.

No disorder of touch, muscular sense, stereognosis, or symbolia.

The reflexes were during the whole course rather brisk, but equal on both sides. No contracture.

This patient sought admission to the clinic owing to stenosis of the œsophagus, which was probably due to cancer; the paresis did not inconvenience him at all. That the paresis was due to a cerebral lesion seems beyond doubt; and being so limited, but at the same time so intense, it must have been situated in the cortex or in its immediate neighbourhood, where it destroyed the centres of the hand and fingers.

As regards the nature of the lesion, metastases are excluded on account of the course of the attack. The almost sudden improvement of the symptoms, setting in as late as two months after the attack, suggests the suspicion of a functional disorder, but the patient's whole manner argued against that supposition, and no other symptoms of hysteria or neurosis could be ascertained; it must be admitted that his motor and sensory disorders corresponded more closely to an organic than to an hysterical paresis.

The strongest argument against a vascular lesion is that improvement set in so late and became so complete; and while the patient was in hospital some changes for the worse were also observed. It is possible that the patient may have suffered, not from a vascular lesion, but from a monoplegia "sine materia" such as is sometimes seen in cases of cancer, general stasis, &c.

This is, of course, a mere conjecture, which does not influence the significance of the case from the symptomatological point of view, since the nature of cortical lesion is unquestionable. The development of the sensory-motor paralysis was interesting; first paresis with a diminished sensibility to touch and pain, and of the muscular sense; later paresis, but no disorder of sensibility except a defect of muscular sense in the little finger; and, finally, pure motor paresis, without even astereognosis, defect in the appreciation of position or of orientation of active movements. Hence it may be inferred that, in cases of cortical sensory-motor paralysis, the sensibility may be wholly restored, but that the cutaneous sensibilities recover more quickly than the muscular sense, and that the latter may recover at different rates in different fingers.

Consequently, a sensory-motor monoplegia may develop from a purely motor paresis. This case completes the two preceding ones, in which there was at first a purely motor paralysis, to which sensory symptoms afterwards were added.

(c) *Cases with Sensory Symptoms* (*Cases XIX-XXII*).

CASE XIX.—L. E. U., aged 63, formerly gamekeeper.

No history of alcoholic abuse or of syphilis.

In the autumn of 1904 apoplexy; the patient fell suddenly, without complete loss of consciousness; paralysis of the left arm and leg, asymmetry of the face; severe headache on the right side. In the evening of the same day the asymmetry of the face had diminished; the power of the leg returned within three weeks, and after a month the patient was able to get about. The recovery of the arm was slower, but the details on this point could not be obtained, except that the patient noticed a certain degree of awkwardness of the fingers and diminished sensibility of the finger-tips after the gradual return of the strength—for instance, he had great difficulty in buttoning his clothes.

Since the spring of 1905 he had been greatly troubled with symptoms of cardiac insufficiency as well as signs of cirrhosis of the liver, this being the cause of his admission to the medical clinic on December 27, 1905.

December 27.—Marked arterio-sclerosis, myocarditis, anasarca, ascites. The power of the left arm, hand, and fingers good; reflexes of the forearm increased on both sides, the other reflexes normal. The muscular sense of the finger-, wrist-, and elbow-joints uncertain, but no noteworthy defect at the shoulder-joint. The finger-finger and finger-nose tests are badly performed with the left hand, finer movements are very badly executed, even when the patient watches them. Stereognostic perception uncertain. Tactile sensation reduced on the terminal phalanges.

December 31.—At 8 30 p.m. the patient was seized with general convulsions, lasting for a few minutes. Conjugate deviation of the eyes towards the right; unconscious. Pupils large, reaction present immediately after the attack. Intellect then clear. No paresis.

January 9, 1906.—No hemianopsia. Cranial nerves normal.

Coarse power of the arms and legs good and equal on both sides. Movements unaffected. The voluntary movements of the arms, especially of the left, were tremulous. The movements of the left arm and hand, especially finer ones, are carried out in an awkward manner even when the patient watches them The co-ordination tests are performed less well with the left hand. When the left hand is raised it remains for a little time in the new position, unlike the right.

The perception of a touch of the camel's-hair brush is uncertain on the distal phalanges of the left hand, otherwise accurate.

Sense of pain, as tested with head and point of a pin, everywhere somewhat uncertain, but distinctly reduced on the left hand and arm.

Stereognosis defective in the left hand. Muscular sense uncertain in the joints of the left fingers, wrist, and elbow. The same applies to the joints of the left foot and toes, but not to the knee.

March 16.—No evident paresis of the left hand and arm. The same cataleptiform condition remains. Does not feel cotton-wool on the terminal phalanges. A certain amount of uncertainty in the appreciation of pain on the hands and feet of both sides.

Great reduction of muscular sense in the left finger- and wrist-joints, only extreme movements being correctly interpreted. At this time a change for the worse in the patient's general condition set in, and he became so drowsy that a closer examination could not be made.

April 5.—Stuperose. April 7.—Death.

Post-mortem (autopsy by Dr. Vestberg): Ascites, hydrothorax, hydropericardium, œdema pulmonum, myocarditis, cirrhosis hepatis (varices œsophagi).

The dura mater normally tense; the soft membranes, as a whole, somewhat œdematous and thickened, especially over the right hemisphere. In the neighbourhood of the fossa Sylvii a thickening of the outer layer of the soft membranes, sharply defined and of a brownish-yellow tint. Beneath this in the posterior part of the fossa the subarachnoid meshwork was increased, showing great cyst-like spaces filled with a clear fluid. This cystic meshwork occupied an area of about the size of a walnut, due to a depression of the brain substance at this place. The region above this area showed a hyperæmic zone, extending from the third frontal to the parietal convolutions. The arteria fossæ Sylvii was patent to the anterior border of the depression in the posterior part of the Sylvian fossa. The depressed area was 5 cm. long, 3 cm. broad, and about 2 cm. deep, and extended to 5 cm. from the occipital lobe. It comprised the gyrus temporalis superior, the posterior part of medius, and the posterior part of parietalis inferior. The surface of the brain was smooth, and the depression covered by cortex.

On frontal section the area was found to occupy nearly the whole of the inferior parietal, the supramarginal as well as the angular gyrus, and a wedge-shaped process almost reached the upper border of the posterior horn. Behind the angular gyrus the area suddenly became narrower, but could be followed almost to the occipital pole as a narrow strip along the sulcus occipitalis transversus. The cortex over the area was atrophic, measuring, as a rule, not more than 1 mm. to 2 mm. in thickness, and being here and there quite destroyed. The ventricles were intact; the basal arteries were thickened.

A microscopical examination of the lower portion of the centro-parietal region was made by the author in serial sections. After hardening in Müller's fluid and embedding in celloidin, sections of 30 to 35 μ in thickness were obtained; the methods of staining employed were those of Van Gieson, Weigert-

Pal (with alum-carmine), nigrosin, and Kulschitsky's hæmatoxylin; every fifth section was stained by one of these methods. The piece of brain examined in this way was about 6 cm. in length; it extended from within 6 cm. of the frontal pole to 7·5 cm. of the occipital pole, and included the white matter beneath the central convolution for at least 1 cm. beyond the lesion; it contained the lower three-fourths of the central convolutions, the posterior part of the parietal lobe, and thus certainly included the centres for the head and arm, and possibly part of the centre for the leg. On microscopical examination the lesion, which was undoubtedly the result of an old softening, was found to be larger than it appeared to the naked eye. The following parts, in addition to the parietal convolutions, were affected: The gyrus centralis posterior, a narrow strip of the insula along the fossa of Sylvius, the gyrus centralis anterior, and the gyrus frontalis inferior. Almost everywhere the lesion involved the white matter immediately beneath the cortex, but the cortex itself was affected in very few places. Its floor was also fairly sharply limited. In addition to the destruction of these parts it produced extensive secondary degeneration, which in

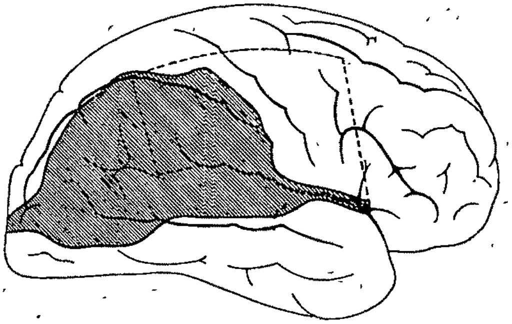

FIG. 6.

Weigert-Pal-stained secretions could be followed far beyond it. In many places the cortex beyond the lesion was atrophic.

The inferior parietal gyrus was completely destroyed. Further, the lesion extended to the lower portion of gyrus parietalis superior, and, as already mentioned, affected the posterior central gyrus directly. The destructive changes found in the latter gyrus were, shortly, as follows: Along the Sylvian fossa the lesion involved the anterior half of the gyrus, but elsewhere only its posterior half, destroying chiefly the subcortex; 1 cm. above the fossa Sylvii it extended from the sulcus post-centralis subcortically towards the vertex of the convolution in its mesial segment (consequently corresponding to the centre for the arm), then it diminished in extent, but, although much reduced in size (1 mm. to 2 mm. in diameter), could be followed through the whole of the sections of the posterior central convolution.

The cortex corresponding to the subcortical lesion was everywhere very atrophic, but was completely destroyed only in a small area in the deeper part of sulcus post-centralis. It was difficult to define the extent of the lesion more exactly on account of the secondary degenerations that were present.

It is easy to understand that a lesion of this extent must have given rise to considerable secondary degenerations, though they could not be accurately traced in Weigert-Pal preparations. Distinct changes of the medullary radiation could be seen in the lowermost portion of the gyrus centralis posterior; the colour of its posterior half was much paler than normal or than when compared with that of the anterior half in Weigert sections. Further, in the posterior half, in addition to the well-preserved fibres, a great number of medullated sheaths were found showing varicosities which were almost entirely absent in the anterior half. Moreover the cortex of the posterior half contained small groups of degenerated ganglion-cells. Higher up the condition was considerably altered owing to the direct encroachment of the lesion upon the convolution, and still more to the secondary degeneration and atrophy, which was here much more intense. The medulla was hardly more than 1 mm. broad, and contained well-preserved fibres only in the anterior half. The cortex of the posterior part of the convolution was greatly atrophied, that of the anterior half much better preserved; here in the cortex as well as in the white matter only small atrophic or degenerative changes could be seen. Judging from their course the intact fibres of the anterior half of the convolution probably represented tracts coming from the depth of the hemisphere.

This marked degree of atrophy of the white matter as well as of the cortex in the posterior portions of the gyrus centralis posterior could be traced through the whole length of the convolution as far as this could be followed in the series of sections, but became less marked higher up in the cortex, and was insignificant towards the upper part of the middle segment. In addition to continuation of the parietal lesion forwards along the Sylvian fossa, there were a few small hæmorrhages, not exceeding 1 mm. in diameter, in the gyrus centralis anterior and at the bottom of the central fissure.

Below, the lesion extended only within the parietal lobe beyond the medullary radiation of the gyrus. The deeper lying portions of the white matter of the frontal and anterior central convolutions showed no signs of alteration. Secondary changes were, however, present beneath the posterior central convolution, and to a still higher degree underneath the parietal convolutions, the deeper radiation being here more purely stained with Weigert-Pal.

Thus the pathological changes consisted of a lesion including, besides parts of the temporal lobe, the lower part of the parietal lobe and the posterior half of the gyrus centralis posterior, especially in its middle third. There was in addition an extensive and intense atrophy of the posterior part of the white matter of the gyrus centralis posterior as well as of the cortex, and in the middle portion a moderate atrophy of the anterior half of the same gyrus.

In spite of the short and incomplete history, this case is of great interest on account of the nature of the sensory monoplegia. The symptoms were a very slight lowering of the cutaneous senses; obvious disorder in the sense of position and passive movement in the left arm; and ataxia and awkwardness in finer movements associated with well-preserved gross movements in the arm.

The patient had a hemiplegic stroke fifteen months earlier, which produced these symptoms. The generalized fit that occurred when he was under observation did not produce any further symptoms, except perhaps a trace of catalepsy and some degree of bluntness of the sense of pain. I must, however, emphasize that, on the patient's admission, attention was directed chiefly to his circulatory disturbances, and that slight catalepsy might easily have escaped attention. The same is the case with the sense of pain, but the defect may have been due to other causes, especially the peripheral arterio-sclerosis.

The other symptoms of lost function in this case are, however, to be considered as due to the lesion that occurred a year and a half before death. Considering the constancy of the symptoms, the case is to be regarded as a residuary sensory monoplegia.

That the power of coarse movements was not diminished is fully explained by the fact that the gyrus centralis anterior, which is regarded as the motor region, was affected by small and doubtless quite unimportant lesions only. The absence of hemianopsia might be accounted for by the fact that the visual tract was damaged to a small degree only.

The greatest interest, however, in this case lay in the disorders of sensibility. Cutaneous sensibility was unaffected in the face, but somewhat diminished in the left arm, in which grave disorders of the muscular sense were also present. The lesion was very small in the lowermost portion of the gyrus centralis posterior, but considerably greater in the middle portion. This unquestionably suggests that the cutaneous sensibility of the face is represented in the gyrus centralis posterior rather than the parietal gyri. And considering the very moderate diminution of cutaneous sensibility in the upper extremity this case argues, it seems to me that, in the front half of the gyrus centralis posterior, there may exist some cortical representation of the cutaneous sensibility of the upper extremity.

This case yields no positive clue for the more accurate localization of muscular sense. As is evident from Campbell's observations, there exists a marked anatomical difference between the anterior and posterior halves of the gyrus centralis posterior, and according to his opinion it might be expected that the "primary" or more uncomplicated functions, amongst which he reckons the simple functions of muscular sense, are localized in the front, the more complicated or "secondary" in its posterior half and in the parietal lobe.

This case gives no support to the view that the sense of position and movement is located in the front half of the gyrus centralis posterior. On the contrary, the intense disorder of the primary functions of the muscular sense argues against the possibility of their being located *only* to the front part of the gyrus centralis posterior, for the slight changes that were met with at this region were only secondary. In the posterior half of this gyrus there is, however, unquestionably a considerable direct destruction that might, with greater probability, have damaged sensibility, and the secondary atrophy and degeneration, which in the anterior half was only sparingly met with, here occupied the whole of the medullary ramification and the greater part of the cortex.

The causes of this secondary degeneration and atrophy, which exceeded the boundaries of the lesion must be sought for in the parietal lesion, with its prolongation towards the gyrus centralis posterior. The remarkable difference in the extent of the degeneration of the fibres of the anterior and posterior halves of the gyrus centralis posterior is undoubtedly due, at least in part, to the circumstance that the intergyral fibres between the gyrus centralis posterior and the parietal gyri mainly go to the posterior part of the former gyrus. It may be, however, that the thalamo-cortical projection paths pass mainly to the anterior edge of the gyrus centralis posterior; but on this my preparations gave no information.

It is very probable that the astereognosis was, in part at least, consequent on the disorders of the primary sensations. But though, in all probability, the intense disorder of muscular sense undoubtedly played an important part in his stereognosis and ataxia, there was, in this case, a fact that may be worthy of note—namely, that the movements were performed in the same manner, whether the patient controlled them with his eyes or not. This points to the disorder being situated in the centre; otherwise it ought to have been compensated to a greater or smaller degree by the sense of sight. Since the motor centres proper were practically unaffected, even microscopically, we have no ground for the assumption of a disorder of "Bewegungsideen" in Munk's sense. It would seem probable that the defect of this function was due to a disturbance at a higher psychical level, but as, on the other hand, it was one-sided, it must necessarily be attributed to his lesion.

Case XX.—P. E. A., aged 71, bricklayer.

In November, 1906, one night he had much pain in the left side of the abdomen with a great deal of meteorism; he obtained considerable relief after passing flatus. He was in bed a couple of months, as the pain and flatulence returned as soon as he tried to get up. He has not been able to determine any connexion between the meals and the onset of the pains. There was no sickness or vomiting. In January he was able to leave his bed; but one day, towards the end of this month, the abdominal pains returned, with pain in the left arm and hand, which became cold and white. The pains also radiated towards the left side of the head. The aching disappeared as soon as the patient kept quiet, but has since then constantly returned as soon as the arm has been moved, and has gradually increased in intensity. Between the attacks of pain he had paræsthesia in the arm and hand, increased on moving.

The patient was admitted to the medical clinic on February 23, 1907, when the following condition was found: Subcutaneous fat and muscles badly developed. Cachectic appearance. General condition bad.

Apex-beat not palpable; dulness to 1½ cm. outside the left nipple line, towards the right uncertain. Second pulmonary and still more second aortic sound accentuated. No murmurs. The pulsations of the axillary artery were more strongly felt on the left side than on the right, but on the left side they could be felt only in the uppermost portion of arteria brachialis; below this level even the artery itself was not palpable. The plethysmograph showed the blood supply of the arm to be very bad. With the tonometer of Landergren the pressure on the right side was 120, on the left not measurable. Gartner 60 on the right, 30—35 on the left. Peripheral arteries on the whole somewhat rigid.

Lungs: Emphysema, bronchitis. Urine: No albumin, no sugar.

Nervous system: Headache, intellect clear; becomes easily giddy on moving the head. Cranial nerves unaffected. Reflexes normal.

The strength of the left hand (dynamometer) 32, of the right 40; otherwise there is no difference between the right and left arms or legs. After protracted exertions the left arm became cold and white, and severe pains came on, rendering every muscular exertion impossible.

He complained of paræsthesiæ of the whole arm, which increase to pain on exertion. No impairment of tactile, painful, or muscular sensations was found on examination.

March 16.—Apathetic, wandering, refuses to eat, passed urine in the bed.

On March 18, in the evening, the patient complained of bad sight. On the 19th, on examining the eyes, left-sided hemianopsia was detected. Tactile and painful sensibility were much reduced on the whole of the left side. Gross disorientation of the position of the left arm and leg. The muscular sense of the left arm seems totally lost, and that of the left leg at least very much reduced. On both sides inconstant results were obtained on testing the joints of the toes and feet, but I could not decide whether this uncertainty was due to a reduction of the muscular sense or the inattention of the patient. Even minute movements of the right knee and arm were, on the other hand, correctly appreciated when the patient answered at all. No signs of paresis. Reflexes normal.

March 20.—Reflexes of the left arm and leg somewhat increased; a certain degree of rigidity. No paresis beyond what has already been mentioned (examination February 23). Is able to perform isolated movements with all the fingers, but associated movements impossible. Is not even able to hold the dynamometer properly.

March 21: Total apraxia of the arm, a certain degree of catalepsy. No sign of paresis. More detailed examination impossible on account of the mental condition of the patient.

Death on March 22.

Post-mortem (autopsy by Professor Quensel): Aortic aneurysm of syphilitic origin with thrombotic laminæ in the lumen, thrombus in the central portion of the left brachial artery, fibrino-purulent pericarditis.

The brain (examined by the author): The dura mater considerably thickened, with a thin easily removable membrane over the frontal lobe. The left hemisphere normal. A flat depression is seen, 1 cm. in diameter, of a yellowish colour and softer consistency, in the posterior half of the gyrus centralis posterior of the right hemisphere at the boundary between its first and second quarters and in the adjacent part of the parietal lobe; the pia mater covering it was injected and somewhat thickened. There was a larger and deeper depression, about 4·5 cm. in diameter, in the middle part of the parietal lobe, occupying the gyri parietalis superior, inferior, and supramarginalis. Towards the middle line the diseased area reached to 1 cm. from the sulcus longitudinalis and forwards to about 3 cm. to 4 cm. behind the sulcus centralis. The anterior boundary was, however, rather diffuse, and the portion of the brain lying between this and the former area was also softer than normal. The area extended posteriorly to 3·5 cm. in front of the occipital lobe. The pia mater covering this part was also much injected and a little thickened.

In the posterior part of the middle frontal convolution as well as in the adjacent part of the gyrus centralis anterior at the boundary, between its middle and lower third, there was an area 1½ cm. in diameter showing similar changes.

On frontal sections of the hardened brain the posterior focus was seen to send a wedge-shaped prolongation downwards almost to the ventricle and forwards towards the area in the posterior central gyrus; macroscopically one cannot, however, make out with certainty whether the affected areas are connected with one another. It seems as if the area in the frontal lobe involved only the cortex and the superficial part of the white matter.

Basal arteries considerably thickened.

Microscopically the membranes over the large parietal softening were injected and thickened, the meshwork was filled with endothelial cells. A large number of veins, and also some arteries, contained fresh thrombi. The brain substance surrounding the arteries and veins, whether thrombosed or not, was infiltrated with leucocytes. The same condition of the vessels was found in the substance of the brain.

The nervous elements within the area affected were destroyed. The neuroglia was in some places œdematous, in others showed signs of breaking up, but was on the whole not grossly affected. Degenerations beyond the softened area could not be established by the staining methods employed. The part of the right hemisphere examined in serial section comprised the whole of the central convolutions as well as the posterior part of the frontal lobe and the anterior half of the parietal lobe; the sections end at about the middle of the affected region.

On the serial sections it became evident, as was to be expected from the macroscopical examination, that the affected parts in the parietal lobe and in the gyrus centralis posterior were connected with each other. The posterior half of the second fourth from above of the posterior central convolution was affected by the softening, which destroyed the cortex as well as white substance forwards to sulcus centralis, but did not encroach upon gyrus centralis anterior. In the peripheral lobe the softening occupied almost the whole of the gyri parietales superior and inferior, with the exception of a strip 1 cm. broad along the sulcus longitudinalis; it reached downwards to the gyrus supramarginalis, a part of which was also involved. On the other hand, the angular gyrus was almost free from destruction.

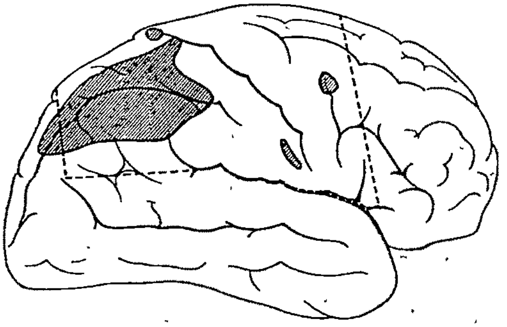

FIG. 7.

In addition, a cortical softening not quite ½ cm. in diameter was found in the immediate neighbourhood of the margo falcata in the posterior wall of the sulcus centralis, and in the corresponding parts of the anterior wall a few small cortical hæmorrhages were found. This small lesion has no connexion with the larger parietal softening.

Finally, in the lower portion of the gyrus centralis posterior there was a lesion 1·5 cm. long and 3—4 cm. broad, situated 5 mm. beneath the cortex layer of the anterior border. It is evident that this is of older date. The posterior central gyrus was otherwise intact.

In the gyrus centralis anterior only minute cortical hæmorrhages were found. The lesion in the gyrus frontalis medius previously mentioned constituted another softening ½ cm. in diameter situated in the anterior part of the convolution; this lesion measured at its centre 2 cm. in diameter on the surface, and sent a wedge-shaped prolongation 2 cm. in length to the deeper parts between the middle frontal and the antérior central convolutions.

In this case there was, in the first place, intermittent claudication of the left arm, evidently due to a thrombosis in the arteria brachialis with the symptoms usual in that disease, but without any disorders of sensibility, excepting the pain connected with his attacks.

I do not intend, however, to dwell on that. Afterwards there supervened an infective process, that caused purulent pericarditis and a softening in the right parietal lobe, that produced hemianopsia and some disorders of sensibility, but no real paresis.

That motility was unaffected by the lesion is explained by the fact that the latter did not extend to the gyrus centralis anterior. The hemianopsia was due to destruction of the visual tract.

The chief interest that the case offered was the disorder of sensibility. This was evidently an early symptom of the lesion and was far more extensive than that which resulted from the lesion itself, especially when, as in this case, the lesion is inflammatory.

Some facts, however, speak in favour of the possibility that in this case distant influence may be neglected. In the first place the reflexes were, even at the onset, somewhat increased. Further motility was unaffected, though the lesion approached to within half the breadth of the gyrus centralis posterior of the motor zone.

Whether the part of the gyrus centralis posterior between the lesion and the motor zone was indirectly affected, by diaschisis, cannot be decided; nor can it be settled whether, in this case, the loss of sensibility was due to the injury of the parietal gyri or to that of the gyrus centralis posterior, though its position within the middle and upper portions of the gyrus centralis posterior agrees with the disorder of the muscular sense in both arm and leg, but it does not explain the affection of cutaneous sensibility that extended even to the face. I do not venture to draw any conclusion on this point, but distant influence seems the most probable explanation.

I wish to point out that, in spite of intense disorders of the muscular sense, the power of isolated finger movements was preserved. That the associated movements were poorly performed is easily accounted for by the grave disorder of the muscular sense. The apraxia that finally supervened, or rather the utter inability to use the arm in spite of well-preserved coarse movements, could not be closely observed owing to the patient's physical state. The reflexes were here a little increased on the hemianopic side even at the onset. There was also a certain degree of cataleptic tension in the whole arm, which indicated the existence of a trace of early muscular rigidity.

CASE XXI.—E. A., aged 23, labourer.

The parents were alive and healthy. The patient was the youngest but one of eight brothers and sisters. Three of these—the first, fourth and eighth—died at the age of 10 months, 3 years, and 11 years respectively, from illnesses not known to the patient. One brother suffered from epilepsy. The patient had had no illness which could account for his present cerebral disease. Seven months before admission cardiac disease was accidentally discovered.

Since the middle of September, 1908, he had been troubled with headache, which at first was localized above the right eye, but afterwards to the right temporal region and to the vertex of the head; during the last few days before admission it was also situated in the right side occipital region. The headache, which for a short time was severest during night, had gradually increased. It came on in attacks lasting, as a rule, for a couple of hours, at first with intervals of two to three days, but during the last week before admission occurred with only short remissions, of about one hour, between the attacks.

For the last two weeks the patient had been troubled with vomiting, which had occurred partly in connexion with severe attacks of headache, and partly in connexion with indigestion. During the last three or four days he had been sick two or three times daily.

For about one month the patient had been troubled with giddiness, especially on bending forwards, and a feeling of uncertainty in balancing the body when walking, and the gait was somewhat staggering. One week before admission momentary diplopia occurred, and he saw his brother with two heads. Once also, a week before admission, he was unable to commence the act of micturition. Bowels sluggish since the beginning of the illness.

No diminution of sight observed; he was able to read easily some days before admission.

The general condition had grown considerably worse since the beginning of the illness; at first the patient was at times able to work, and even two weeks before admission he could remain out of the bed for a little while, but since then he has been confined to his bed.

His weight at the beginning of the summer was 70 kg., on admission 55·8 kg. He had grown somewhat dull and his memory had been getting gradually worse since the beginning of the illness. He was admitted to the medical clinic on October 31, 1908.

Condition on admission: Lacks initiative and answers questions slowly; no real blunting of the intellect can be made out, the patient, on the contrary, being very attentive, and on this account easy to examine. Respiration 16; pulse 64 in the minute.

He is pale and thin. Complains of headache, which sometimes increases to violent pain. There is no difference on percussing the right and the left parietal region. During the painful paroxysms the patient is tender to percussion over the right temporal bone and behind the right ear, and on turning and bending the head pain is also elicited. These symptoms disappear between the paroxysms of pain.

Taste normal. The visual fields on coarse examination unconstricted, at least no hemianopsia. Double optic neuritis, most marked on the right side. The movements of the eyes and the pupils normal. Sensory and motor functions as well as reflexes normal. The left sulcus naso-labialis is less marked than the right one. Closes the left eyelid less quickly and less effectually than the right one. Slight tremor of the eyelids on both sides. He is able to hear a watch at a distance of one meter on the right side, at a somewhat greater distance than on the left. No difference in the perception of taste on the two sides of the tongue, and this applies to its anterior as well as to its more basal part. The left arch of the palate stands possibly somewhat lower, and is not raised as high as the right on moving the palate. Glossopharyngeal intact. The tongue deviates a little towards the left.

No signs of paresis. Tremor of the fingers.

Sensibility to pain and touch normal (examined with cotton-wool and pin). To the finger-nose or finger-finger test there is no ataxy in the hands; the knee-heel test is also well performed. Romberg's sign is slightly positive. He does not separate his legs unduly when he walks, but he staggers somewhat when the eyes are shut.

Arm reflexes normal, patellar reflexes brisk, especially on the right side. Achillis-tendon reflexes increased, slight ankle clonus on both sides. The abdominal reflexes equal on both sides, cremaster reflex brisker on the right side.

November 2.—Lumbar puncture, pressure 330 mm.; the fluid contains 1-2 lymphoid corpuscles in every 5 cubic mm. The facial paresis increased; no disturbance of the muscular sense of the fingers and toes.

November 5.—After an attack of severe headache, dynamometer right hand 35, left 30. The power of isolated and complicated movements of the fingers good on both sides (could fasten a button, &c.).

Muscular sense: The sense of position and movement in the fingers and wrists is considerably diminished and some uncertainty in the elbow-joint on the left side for minimal movements. In the joints of the toes on the left side there is some diminution of the muscular sense. Touch intact in the hand, though possibly some diminution laterally on the left foot (cotton-wool).

Orientation in space: Right hand recording, iliac level, an average error of 6·6 cm. in caudal and of 2·6 cm. distal direction; xiphoid level, 6·8 in caudal and 2·3 cm. in proximal or distal direction. Left hand recording, iliac level, error 3·5 cm. in caudal and 1·5 cm. in proximal direction; xiphoid level, 1·5 cm. in kephal or caudal and 1·5 cm. in proximal direction.

The perimeter examination showed an inconsiderable concentric limitation of the visual fields on both sides, but no hemianopsia.

November 6.—Sensibility unaffected, motility as before.

November 7.—Puncture of the brain over the middle frontal convolution and over the temporal lobe on the right side. Only a small quantity of blood was obtained.

November 10.—After being considerably better for the last two days the patient is apathetic again to-day. He has headache, vomiting, and stiffness in the neck, as well as tenderness over the upper cervical vertebræ. The sensibility shows the same disorder as on November 5—i.e., an almost isolated, but obvious, disturbance of the muscular sense.

November 11.—Smell and taste unaffected; right corneal reflex increased; no hemianopsia. The left pupil is smaller than the right, but both react.

November 12.—Ptosis of the right eye, the pupil being larger on this side. Nearly complete absence of corneal reflex. The movements of the right eye reduced, especially of internal rectus. The examination of the visual fields, which could be made only very approximately, seem to indicate left-sided hemianopsia. No paresis in the arm.

In the afternoon twitchings in the left shoulder (raising). At 10.30 p.m. unconscious. Pulse 180—200, respiration 7.

Lumbar puncture pressure 500 mm.: 6 c.c. were drawn off, the pressure sinking to 170 mm. Exitus at 11 p.m.

Autopsy by Professor Quensel (only an abstract of the report on the brain being here given): The tension of the dura mater is considerably increased, the thickness is normal, the inner surface dry with increased blood supply. Round about the punctures there are small calcareous particles on the inner side of the dura.

On the convex surface of the brain the gyri are flattened on both sides to about the same degree, the sulci being almost effaced. The right lobus temporalis considerably enlarged and much broader than normal, and of fluctuating consistence. The lateral ventricles empty and not enlarged.

The brain was hardened in formaline, and, after the hardening, was examined by the author in frontal sections. Pathological changes were not found until a frontal section, lying 7 cm. behind the frontal pole and 2·5 cm. behind the apex of the lobus temporalis, was reached. In the external part of the medullary substance of the first temporal convolution in this section an area was seen 1¼ cm long and ¾ cm. broad with the greatest extension in horizontal direction, of somewhat softer consistency and of a yellowish colour. This area reached the cortex but did not involve it: 7·5 and 8 cm. behind the frontal pole the discoloured area was 2½ cm. long and 1 cm. broad. The colour was here more yellowish-red. In the centre of this discoloured area, around a longitudinally cut blood-vessel, a hæmorrhage was seen with its largest diameter in the horizontal direction, 8·5 cm. behind the pole of the frontal lobe, there was a hæmorrhage 4 cm. long and 1·4 cm. broad, with the greatest extension in the horizontal direction, occupying the white substance of the superior temporal gyrus, which was here considerably enlarged, as well as the central part of the lobus temporalis. Outside the area occupied by the hæmorrhage itself the white substance showed a yellowish-red discoloration, and had a somewhat softer consistency over an area about 5 mm. in width. The softening reached the cortex of the temporal lobe in the first temporal gyrus, and extended to about ½ cm. from the cortex in the second temporal convolution, sending a narrow prolongation ½ cm. up between the cortex of the insula and the claustrum. The softening was separated from the cornu inferius by a portion of macroscopically normal brain substance 8 mm. wide. The nucleus lentiformis was not involved; 9·5 cm. behind the anterior pole of the lobus frontalis the hæmorrhage is 1·5 cm. broad and 5 cm. long, otherwise as before, 10·5 cm. behind the frontal pole the white substance of the temporal lobe is almost completely occupied by the hæmorrhage, which was here 1·8 cm. broad and 5 cm. long. Here the surrounding softening had also involved the cortex of the upper convolution, and had extended laterally as far as the cortex. On the mesial aspect the softening reached to 4 mm. from the cornu inferius and 5 cm. from the nucleus caudatus, and had evidently destroyed part of the projectory tracts of the lower half of the gyrus supramarginalis; 12 cm. behind the frontal pole the hæmorrhage had an extension of 6 cm. length and 3 cm. breadth. Upwards and outwards it was bounded by macroscopically unaltered cortex. Internally the hæmorrhage was bordered by a softening more than 1 cm. broad, which reached the lateral ventricle. The ventricle was empty. The lesion occupied on this section the upper and middle temporal gyrus, as well as a narrow strip of the gyrus angularis; 13 and 14 cm. behind the apex of the lobus frontalis, the hæmorrhage was considerably less extensive, being only 2 and 1·5 cm. long and 0·8 and 0·5 cm. broad. It was still largest in the horizontal direction. It was situated to the outer side of the ventricle, and separated from it by an area 4 cm. broad, which was softened down to the tapetum; 16 cm. behind the frontal pole, on the lateral side of the cornu posterius, there was a softening of about 1 cm. diameter. 17 cm. behind the frontal pole the surface of the section appeared normal.

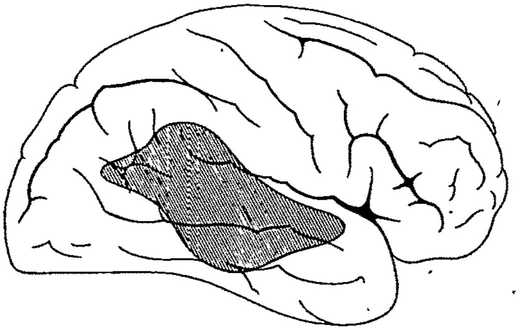

FIG. 8.

To the naked eye there thus appeared to be a hæmorrhage within the lobus temporalis 7, 6, and 3 cm. respectively in sagittal, frontal, and vertical extent which was surrounded by an extensive area of softening. The parts which were destroyed by it and the surrounding softening were the first and a great portion of the second temporal gyrus, the lower half of the gyrus supramarginalis and angularis, as well as a small part of the insula. Macroscopical examination did not reveal any tumour, and the microscopical examination of several pieces from the brain-substance surrounding the hæmorrhage showed a softening only

Microscopical examination was made of the portions of the gyrus centralis, nearest the lesion—i.e., their lower third—also of the gyrus supramarginalis and of the gyrus angularis and of the insula; the superior, middle, and inferior part of the basal ganglia and capsula interna were also examined microscopically. The gyri centrales and the basal ganglia, as well as the capsule, showed no changes. At the bottom of the fossa Sylvii there was a small hæmorrhage, surrounded by a narrow area of softening, which also involved the claustrum to a small extent.

The softening in the gyrus supramarginalis was more extensive when examined with the microscope than it appeared to be to the naked eye; the projection tracts of almost the whole of its lower half were destroyed. The nucleus caudatus was unaffected, as well as the upper part of gyrus supramarginalis and the gyrus angularis.

The previous history pointed directly to a tumour cerebri with severe general symptoms and rapid progress. The symptoms in the order in which they appeared were: (1) headache, coming on in attacks with always shortening intervals; (2) giddiness, especially when bending forwards, and a staggering gait; (3) vomiting, partly in connexion with attacks of headache and partly in connexion with indigestion; (4) momentary diplopia and sphincter trouble. In spite of a marked optic neuritis at the time of admission, the sight was so little affected that the patient was able to read till shortly before admission. To this was added loss of strength and a certain amount of psychical impairment, and during the last course of the illness diminished muscular sense of the arm and foot, and paresis of the face, the oculo-motor and trigeminal nerves.

Post-mortem there were, however, no signs of a tumour, and only a large hæmorrhage was found. The presence of the hæmorrhage from the time of onset can be excluded by the clinical course of the case. On the other hand, there may have been a tumour with a secondary hæmorrhage, for it is well known that in certain cerebral tumours, especially gliomata, it may be very difficult to discover the new growth under such conditions. Thus the fact that post-mortem no tumour was found cannot be regarded as of great importance, as the illness presented all the characteristics of a cerebral tumour; the diagnosis tumour must consequently be considered justified.

Evidently the original lesion was less extensive than that found after death; one cannot judge of its actual dimensions, and consequently of its situation within the area involved by the lesion found after death. But, as the middle and posterior part of the temporal lobe were destroyed by hæmorrhage and areas of softening, it seems clear that the seat of the tumour was probably here, and consequently it may have affected the centre for the hand from behind and below. This view is supported by the fact that hemianopsia was discovered before any signs of paresis of the arm appeared.

The fact that the sensory disturbance was limited to a defect of muscular sense in the arm and foot seems to me of special interest. These sensory symptoms were associated with attacks of grave general cerebral symptoms similar to those in a case described by Binswanger. The case in this respect corresponds to Case XVII. with the difference that in Case XVII. the lesion exercised its influence from in front, whereas in this case it probably affected the sensory zone from behind and below without directly infringing upon it, as the histological examination showed that the internal capsule and the central convolutions were unaffected, and only the lower part of the parietal lobe contained an area of softening.

This case thus proves that a lesion which affects the centre for the hand from behind and below may give rise to disturbances of muscular sensibility and to orientation in space without necessarily affecting motility; further, that the muscular sense may be more readily affected by diaschisis than cutaneous sensibility.

Further, this case is of great interest as it presented symptoms of a focal lesion in a region as "dumb" as the right temporal lobe. The grounds upon which the diagnosis was made were: The marked general symptoms associated with very slight local symptoms indicated that the tumour was situated in a mute region; the pains in the right side of the head and a slight paresis of the left facial and hypoglossal nerves seemed to indicate that the tumour had to be looked for in the neighbourhood of the lower part of the motor zone; there was no reason to assume that the tumour lay in the frontal lobe, as the slight staggering which the patient showed could not be interpreted as a frontal ataxia when his general condition was taken into account; nor was his psychical condition such as one would expect to find in the case of a frontal tumour; further, the absence of any disturbance of the nerves of smell on the right side argued against a tumour in the lower portion of the right frontal lobe. On the other hand, the fact that, at a relatively early stage of the observation, sensory disturbances of a transient nature only, it is time, were observed in the hand, when its motility was not affected, was a reason for assuming the lesion was situated behind the motor zone. Such a condition, as may be gathered from the literature, is common in the case of lesions situated behind the motor zone, and in agreement with it stand my cases Nos. XIX and XX. On the other hand, the absence of hemianopsia in the presence of motor paresis of the face was an argument against a parietal tumour. A lesion situated behind the motor zone ought not as a matter of fact to have shown its effect only upon the lower third of this area. For this reason it was not likely that the lesion was situated in the parietal lobe, but probably exercised its influence upon the motor zone from behind and below—i.e., it was situated in the posterior part of the temporal lobe. The basal headache might be in favour of a tumour localized to the base of the brain, but a more definite argument for this localization was the symptoms of loss of function observed on November 11 in the trigeminal and oculo-motor nerves. Lastly, the hemianopsia unassociated with loss of power in the arm strengthened the supposition that it was the posterior part of the temporal lobe that was the seat of the tumour.

CASE XXII.—K. E., aged 64, housewife, [Abstract of notes by Mr. Möller.]

No previous illness of interest. On January 27, 1903, a sudden attack of twitching of the left arm and the left side of the face which began in the hand, passed up the arm and involved the cheek: the twitchings were said to have been felt only, not seen. She had five such attacks on that day. They were not attended by headache, vomiting or giddiness, nor by paræsthesiæ or pain. In the evening of the same day weakness of the left arm; on the next day she was unable to perform any finer work, such as knitting, &c., and could not fasten a button with her left hand. Her condition remained unaltered till two days before admission, when the left leg also felt weak. Nevertheless, on February 4 she walked to the hospital, a distance of 15 kilometres. She had been up and about the whole time and partly working.

February 5.—On admission mental state natural. Subjectively she feels well, no headache or giddiness. A feeling of weakness of the left arm and, to a lesser degree, also of the left leg. No hemianopsia; fundus oculi normal on both sides.

Tactile sensibility in the area supplied by nervus trigeminus somewhat uncertain on the left side (hard touch with a camel's-hair brush). She could differentiate between the head and point of a pin, but describes the impressions as being weaker than on the right side.

February 9 to 12.—The general condition of the patient much the same. Only complained one day of aching in the left foot. Slight concentric limitation of the field of vision. The left corner of the mouth hangs a little lower than the right; the tongue deviates somewhat to the left. The left arm immobile, flaccid, cyanotic, œdematous; it feels warmer than the right one but is very flaccid.

From about the knee downwards she does not feel the camel's-hair brush or a pin-prick; above the knee subjective diminution. On the left hand impairment of tactile and painful sensation to examination, subjectively on the whole arm.

On February 11, as the patient was sitting in a chair, she suddenly fell without losing consciousness. There was no immediate paralysis, but on the following day the left arm was immobile and the left leg weak.

February 13.—During the night severe headache. In the morning of February 13 a fit lasting twenty minutes; the patient did not lose consciousness. During the attack convulsive movements of the left arm, leg, and left side of the face, and the head moved to the left side. The convulsions were short and so violent that the patient shook the bed; the fits came on three times that day, but lasted only a few minutes. The left leg was paretic; the left eyelid was less tightly closed than the right one, and the wrinkles of the left side of the forehead were almost obliterated. Distinct paresis of the lower part of the face; the tongue deviated markedly to the left; left-sided hemianopsia. Speech slurred but intelligible; cranial nerves otherwise unaffected.

Later there were occasionally slow involuntary movements of the left upper limb consisting of abduction of the arm and flexion of the elbow-joint. Range of passive movements natural, but there is a trace of rigidity of the left arm and great limitation of active movements; the arm can only with difficulty be placed on the head. Extension and flexion of the left elbow-joint weaker than that of the right. With the dynamometer no difference as regards the two hands, but a squeeze of the left hand feels weaker. Electrical reactions normal. States that the current feels stronger on the left side.

On the left arm the patient feels a light touch with camel's hair brush down to the wrist; she states that at times a touch feels weaker on the left forearm than on the right. A touch is not felt on the ulnar half of the dorsal aspect of the left hand nor on the palmar aspect to the ulnar side of the middle finger. On other parts of the hand, too, a touch is said to be weaker than on the right. Recognises the difference between the point and head of a pin on the left arm but is somewhat uncertain on the hand. Describes the impressions as weaker on the left side. Localization good.

When the patient is asked to place one of the fingers of the left hand on the nose, this movement is performed in a fumbling and awkward manner. The patient is unable to recognize the direction of passive movements of the joints of the fingers of the left side. A similar degree of uncertainty exists in the left wrist, whereas movements of the elbow-joint are recognized. The patient is unable to distinguish between different objects such as a key and a knife with the left hand, and is unable to knit or fasten a button with it.

Sense of temperature impaired; the patient is only able with difficulty to distinguish between cold and warm water on the left arm and hand. The impressions are said to be weaker on the left side.

The biceps- and triceps-reflexes brisker on the left side.

February 14.—Urine and fæces pass in the bed. Stuperose and does not respond to speech, or recognize her husband. She was unable to swallow in the evening. Death on February 17.

Post-mortem.—On opening the cranium the dura mater was found tense. On the right hemisphere a projecting area yielding more to pressure than other parts of the brain and extending from the fissure of Rolando about 6 cm. in a direction backwards. On incision this area was found to consist of a cavity about 4½ cm. deep containing thin blood as well as a blood-coloured mass posteriorly. The brain substance which formed the bottom and walls of the cavity showed distinct signs of softening. On opening the right lateral ventricle this contained a large quantity of fluid blood, and was much dilated. Immediately behind the centre of the corpus callosum on the right side a yellowish discoloured area of about the size of a sixpenny-piece. On inspecting the brain, blood was seen to emerge from the optic tract. The weight of the brain was 1,360 grm. In the hardened brain the diseased area was found to communicate with the ventricle at one small place.

The case is one of a patient who had repeated fits. The first was preceded by phenomena of irritation, repeated twitches, which were so feeble that they could be felt but not seen, but which were nevertheless extensive. This contrast between the extent and severity of the motor symptoms makes it probable, especially considering the absence of general symptoms, that at this time there was no gross vascular lesion, but merely a disorder of nutrition within the motor zone. Later there was an intense but temporary paralysis, and after a further Jacksonian fit on February 13, a very irregular loss of function—namely, a motor paralysis in face and tongue, a sensory paralysis in hand and fingers, especially of muscular sense, a left-sided hemianopsia, and, finally, a motor paralysis combined with phenomena of irritation in the shoulder- and elbow-joints. Finally, another fit was followed by severe progressive paralysis of the whole of the left side.

From the history it is evident that the large lesion found post-mortem cannot give any further information as to the site of the lesions that occasioned the earlier fits. It is, however, evident that the gyrus centralis anterior—that is, the motor zone—cannot have been involved in the early lesion; and, considering the simultaneous hemianopsia, it is obvious that the lesion of February 13 was situated within the region between the gyrus centralis anterior and the occipital lobe.

It is certain that the lesion found at the post-mortem examination developed or extended considerably after February 13; for symptoms of so moderate intensity and extent as those observed at that time are incompatible with a vascular lesion of that extent.

A full examination of the brain could not be made, as in consequence of a too long treatment with Müller's solution it had become brittle. The case is of value chiefly from the clinical point of view, but it is important that the lesion was demonstrated behind the sulcus Rolandi—that is, behind the motor zone.

It is interesting that the partial distal hypæsthesia was of an ulnar type in this case as well as in Cases XIII., XV., XVI., and the reverse of that found in Case V.

The normal power of localization in spite of the disorders of the uncomplicated cutaneous senses is absolutely at variance with my experience from other cases, but I cannot enter upon that matter, as no further particulars are recorded in the notes made at that examination.

CHAPTER IV.—THE SENSORY IMPAIRMENT DUE TO CORTICAL LESIONS.

§ 1.—*Sensory Impairments in General.*

The sensory impairments following a cerebral lesion have formed the subject of detailed investigations to a much less degree than the disturbances of motility. Their significance to the patient is less ominous; they frequently pass away and, not least, the circumstance that they are much more troublesome to examine, fully explains this neglect. Moreover, it cannot be denied that a minute examination of a case of cerebral hemiplegia with regard to sensory functions may be sorely trying to the patience of the doctor as well as of the patient. Still, even if the statements diverge greatly with regard to many important details, all observers seem to agree on several of the main points. (Aba, Brécy, Dejerine [48], Henschen, Long [118], Monakow [141], Müller, Oppenheim [158], Verger [218, 219].)

Firstly, with regard to total lasting hemianæsthesia, taken in the strictest meaning of the word, it seems certain that it must be very rare if it exists at all, except in association with hysteria; whereas, on the other hand, slighter, incomplete and partial, as well as dissociated sensory defects are common.

I will discuss first the sensory impairment which follows a lesion within the boundaries of the internal capsule, since these are the best known and studied on account of their intensity and wide extent. Out of twenty-six cases of residuary hemiplegia I myself have found evident signs of impairment of superficial, as well as of deep sensibility, in nineteen cases, and in the remaining seven I was unable to exclude changes in sensation on account of the inattentiveness of the patient. Immediately after the onset of the hemiplegia one sometimes finds, according to opinions prevalent in the literature (Müller, Monakow, Verger, Dejerine), a total hemianæsthesia and hemianalgesia of the paralysed side, extending even to the mesial parts of the non-paralysed side (Dejerine), or to the distal parts of the lower extremity of the non-paralysed side (Müller). More often, however, according to the same accounts, it is more common for the anæsthesia to be total and complete, as far as tactile sensation is concerned, whereas painful sensations are not entirely absent on the paralysed side, although a powerful stimulation, or even a summation of stimuli, may be necessary to reach consciousness or to give rise to a reflex movement (Marie and Guillain). Should the patient react to the painful stimulus, it sometimes happens that, although he complains of no real pain, he becomes restless and excited without being able to account for this condition (Müller).

This initial, deep hemianæsthesia coincides in time with the initial limp paralysis, and has been placed side by side with it by Monakow. According to this author, both are due to a functional disjunction of the neuron-chain, a "Kontaktlockerung."

Even when the initial sensory disturbance has been of such intensity, it becomes permanent in an exceedingly small number of cases only excepting where a superposed hysteria gives rise to an anæsthesia.

Hypæsthesia on the Healthy Side.—A slight encroachment of the sensory disturbance upon the central parts of the "healthy" side is not seldom observed in the case of total hemianæsthesiæ (Dejerine). Further, Friedrich Müller in two cases (his cases Nos. 6 and 11) of cerebral hemianæsthesia, neither of which seems to have been of cortical origin, has been able to ascertain the presence of hypæsthesia in the distal parts of the lower extremity of the non-paralysed side, at least with regard to cutaneous sensibility.

In two cases of capsular lesion with hemiplegia and hemianæsthesia (Cases I. and II. of this paper) I have had opportunity to make a similar observation, viz., in Case I. a transient hypæsthesia of the mesial parts of the trunk on the healthy side, as well as a hypæsthesia of the lower extremity of the non-paralysed side remaining for some time, and in Case II. a very moderate hypæsthesia of the sound lower extremity.

Further, I have lately had an opportunity to observe a young patient, aged 30, with an intense hemiplegia, and anæsthesia due to a cerebral embolism. In this case there remained, two weeks after the onset, at the time of my first examination, besides total paralysis of arm and leg, paresis even of the upper part of the face and forehead, as well as of the muscles of the trunk, chiefly on the right side; the strength of the left arm and leg was certainly not appreciably reduced. Besides this, a high degree of anæsthesia was here present, reaching on the trunk 3—4 cm., in the face 1—2 cm. on to the non-paralytic side. There was no anæsthesia of the left foot. Three weeks after the onset the condition remained on the whole unaltered, with the exception that the paresis of the trunk had diminished. The reflexes were unaltered on the left side, increased on the right. Further, in a case of cortical sensory-motor monoplegia (Case XIII.), I have also found a reductions of the muscular sense in the hand of the healthy side.

Fr. Müller considers that this distal hypæsthesia of the non-paralysed side is due to a double cerebral innervation, and his case supports such a supposition; here the paresis had affected the muscles of the trunk on both sides, and the knee-jerk was also exaggerated on the non-paralysed side. To this an objection might, however, be made. In my Case I. the anæsthesia extended partly to the mesial parts of the non-paralysed side of the trunk, partly to the distal parts of the lower extremity of the same side. If the cause of the hypæsthesia were the same with regard to both areas affected, the retrogression of both ought to go on the same lines. This was, however, not the case. With regard to the trunk, the anæsthesia of the mesial parts of the non-paralysed side quickly disappeared. Next the sensibility of the paretic side also quickly improved, and this restoration progressed on the paretic side in a lateral direction This is exactly what one would expect in the case of a bilateral innervation, such as most likely exists with regard to the mobility and sensibility of the trunk.

As far as the lower extremities are concerned, it was the distal part of the non-paralysed leg which was affected by the anæsthesia, both in my Cases I. and II. and in Müller's cases. Consequently it ought to be these that were innervated bilaterally; but here the improvement progresses in a distal direction both in the paretic and non-paretic side—i.e., the parts with double innervation would seem to show less tendency to repair. Such a supposition is very unlikely. My case just mentioned showing a paresis of the trunk and anæsthesia of the mesial portions of the trunk and face on the non-paretic side, but at the same time normal sensibility in the foot, speaks also to a certain degree against the view held by Müller.

The same applies, according to my opinion, to a case like No. XIII. of my series. The bilateral sensory disturbance had in this case affected the upper extremity, too, but here it was the muscular sense of the healthy hand that was affected; and when dealing with the hand, especially with a function as specialized as the muscular sense, all our other experience prevents us from assuming a double cerebral innervation.

A plausible explanation can be found by assuming not a bilateral *innervation* but a bilateral *lesion*, that in the "healthy" hemisphere being of a relatively slight and transient nature. It is evident that in connexion with an apoplexy due to a hæmorrhage or thrombosis, the cerebral circulation might be unfavourably influenced far beyond the real extent of the lesion and by such means might give rise to a diaschisis effect. A process like this is likely to influence sensibility also. If such is the case, it is not unlikely that a disturbance of the central apparatus of this kind would first of all manifest itself in such places as the distal parts of the lower extremities—especially in elderly persons—already physiologically provided with less acute sensibility. In the two cases where Müller observed a distal hypæsthesia on the healthy side, the patients were about 60 years of age, and in the Cases I. and II., in which I myself found the above-mentioned symptom, one was a drinker aged 66, and the other an arterio-sclerotic patient of 74. As a contrast to this stands the last case examined by me, a young patient without any signs of arterio-sclerosis, showing a considerable hypæsthesia of the mesial part of the trunk on the healthy side, but no distal hypæsthesia of the leg. These facts seem to me easy to combine with th[illegible] supposition of a double lesion

The primary anæsthesia is, however, as a [illegible] not complete, and generally less intense in the mesial and proximal than in the lateral and distal parts, and most marked in the extremity, the motility of which is more paralysed—i.e., as a rule, in the arm, especially in the hand. When the disturbances begin to retrogress, which at first usually takes place quickly, the restoration of the function begins in the parts situated nearer the middle line or more proximally.

The sensory disturbances persist a long time in the distal parts, especially of the upper extremity, but may finally disappear even here. The rule is, the more extensive a sensory disturbance the more intense, and *vice versa;* this was demonstrated by my Cases I.—V., in which a sensory disturbance situated in the hand only was not complete, at least as far as cutaneous sensibility was concerned. During the early stage, as well as during the further course, the disturbances behave differently with regard to the different qualities of sensibility. Where tactile sensation is being restored, a process which starts near the middle line proximally and progresses towards parts situated more distally and laterally, one finds according to the localization and intensity of the disorder and according to the length of time since the onset of the injury all quantitative degrees, from insensibility to even a hard touch (as in my Case II.), to slight reduction (as in my Cases IV. and V.), or even subjective diminution of sensation only. Besides this, as pointed out by Verger and Müller, a qualitative disturbance is often found, the patient being unable to recognize the nature of the stimulus.

Under normal conditions, when testing painful sensations quantitatively by means of Thunberg's or Alrutz's algesimeter, one always gets a sensation or touch with less weight than that necessary to elicit pain —i.e., when applying methods suitable for the examination of painful sensations, the liminal intensity of the sense of pain is higher than that of the sense of touch. When the lesion lies in the spinal ganglia (herpes zoster) the disorders of tactile sensation have, at least on the trunk, a greater power of compensation in consequence of an overlapping supply than have disorders of the sense of pain (Petrén and Bergmark). In the case of a cerebral lesion matters are different. The sense of pain, when tested with a pin, may be found intact, or only slightly affected in parts where tactile sensation is highly reduced (see my Cases III. and IV.), just as its restoration, as a rule, takes place quicker (my Case I.), and on the whole the disturbance is less extensive than that of the sense of touch. These rules, however, are far from being without exceptions. Thus Gordon more often found disorders of the sense of pain than of the sense of touch, and in my Case V. the disturbance of painful sensation was more extensive than the hypæsthesia. Further, Müller occasionally observed a certain degree of reduction with regard to faradaic painful stimulation and was able to demonstrate this for months after the onset of the lesion. The disturbances of painful sensation are, however, according to my experience, rarely of any magnitude during the later stages, and a complete analgesia of even the distal parts is very rare.

Hyperalgesia of the Paralysed Side.—Besides the symptoms of lost function, one finds also phenomena of excitation on the part of cutaneous sensibility such as paræsthesiæ or spontaneous pains in the paralysed side (Œdinger [59], Schaffer [183], Henschen). Sometimes also hyperalgesia is found, with summation of stimuli (Friedländer and Schlesinger, Long), so that a touch or a light prick is perceived as real pain, or, at least, is more uncomfortable to the feeling than on the other side (Fr. Müller). As no determination of the liminal values seems to have been made in these cases, it may be of interest to know that in those two cases of capsular lesion where I found similar though not very marked phenomena—in the one (Case V.) a slight degree of hyperalgesia, in the other case (Case I.) after-sensations of a more troublesome nature—the liminal intensity was not below normal, but, on the contrary, was somewhat higher than normal for the superficial sense of pain. In the cortical Case XIII. the liminal value was higher on the hyperalgetic side.

The expression "hyperalgesia" consequently is somewhat improper in these cases, as such a name would imply a lower value of the liminal intensity than normally (Petrén and Carlström). Still, in my cases, the hyperalgesia was very slight only, but it seems to me not without interest to examine cases showing a higher degree of hyperalgesia with regard to this point. I may further add that, in a case of tumour in the thalamus with severe spontaneous pains in the hemiplegic side, I was unable to find any signs of hyperalgesia at all in the course of the ordinary examination with a pin.

Other Forms of Sensory Impairment.—The thermal senses I have not tested in detail, nor do I intend to dwell on their behaviour in cases of central paralysis, the more so as opinions diverge on this point. Fr. Müller, for instance, found them more affected than the common tactile sensation, whereas Verger only rarely found any impairment. Often the sense of heat is, according to Henschen and Fr. Müller, more affected than the sense of cold.

I have here briefly referred only to disorders of the "simple" cutaneous sensations, and their occurrence in capsular lesions and in general. The impairment of the more complicated functions, the power of localization and the muscular sense is, as a rule, still greater, but in order to avoid reiteration, it may be as well lightly to touch upon them in this connexion.

As mentioned in the chapter on methods, I have in my cases made a more thorough examination of the power of localizing a touch. This function may be fully extinguished immediately after the onset of the capsular injury (Fr. Müller), the patient having no perception at all of locality, not even a wrong one. Later on, perception of locality returns—maybe only of a very uncertain kind. Thus Schaffer [184] describes a case of uncomplicated circumscribed capsular lesion where the patient localized a touch on the lip to the leg, &c. Similar cases have also been observed by Oppenheim [158], and an atopognosis of a marked degree, even if not as paradoxical as the above, is mentioned by several authors.

Müller, for instance, relates that after a mechanical stimulation the patient often localized the pain to the extremity as a whole. An atopognosis very irregular as regards its direction has been demonstrated by several authors. In other cases, again, a certain degree of regularity of the atopognosis was noticed, the patient localizing too far proximally, as has been pointed out by Verger [219]. Müller, Schittenhelm and Sandberg found greater disturbances with regard to localization only in cases where the respective cutaneous sensation was itself affected. The rule is, however, that, in connexion with capsular lesions, tactile sensation presents long-lasting disturbance in the distal parts; and here disorders of localization, even severe ones, are, according to my experience, the rule. Such disturbances were found, however, outside the area showing disorders of tactile sensation, a fact which seems to agree well with common experience. To this point also I shall return later.

The deep sense of pressure, to the importance of which especially Strümpell [206] has called attention, may, according to this authority, present grave disorders in connexion with cerebral lesions without any affection of tactile sensation, the patient having no perception of a hard pressure directed against underlying structures—such as muscles, fasciæ, periosteum—although a superficial touch can be perceived. Fr. Müller was unable to prove this, but, on the other hand, the opposite condition was not uncommon—viz., pressure was correctly appreciated while the touch was not felt. My own experience goes in the same direction.

The deep sense of pain (Liepmann [116]) was found absent in a case of infantile hemiplegia, the superficial sense of pain being intact.

According to the almost unanimous opinions expressed in the latest literature, the various functions having reference to the perception of muscular sensation and position are most severely and lastingly affected. Both with regard to the simpler and, above all, the more complicated functions, disturbances often remain for a long time after the restitution of other sensory disorders, thus constituting what Verger called a "hemianæsthesia forme fruste." Stereognosis especially is, as a rule, severely affected. I must, however, point out that one author (Gordon) has arrived at another result; he found disorders of cutaneous sensibility in thirty-two, and of muscular sensibility in seventeen cases, out of thirty-five. Even granted that in several cases the cutaneous disturbances were slight, and did not imply all the qualities, still this result stands alone.

Further, the impairment of muscular sense is most intense and remains longest in the distal parts, but may disappear in very old cases; this applies even to the astereognosis (Long [118], Marie and Guillain). This occurs, however, in very rare exceptions, and a man with an experience of the pathology of the brain such as Monakow [140] even doubts such a possibility.

§ 2.—*Sensory Disturbances after Cortical Lesions.*

Verger has pointed out that the sensory disturbances found after cortical and capsular lesions do not present any differences in principle. This statement I can fully endorse as far as qualitative differences of those sensory functions are concerned, with which I deal in this paper—namely, tactile and painful sensations, power of localization, and perception of passive movements. With regard to frequency and distribution, on the other hand, essential differences occur.

First of all, it appears from the literature that in cases of cortical monoplegia sensory disturbances may be absent. Also in a few cases of old hemiplegia, Long, Marie and Guillain have, as above mentioned, found even the stereognostic function intact, but this probably happens in an exceedingly small number of cases and only when the hemiplegia is of long standing.

When we have to deal with cortical lesions, matters stand differently. Here motor disturbances may take place without simultaneous sensory disturbances, as has been shown by Mills, Monakow, Müller (*see* Chapter V). How common such an isolated motor paralysis may be in connexion with a cortical lesion it is of course impossible to decide. According to my experience it would not seem by any means a rare thing, as in several cases (Cases VI.—XII.) I have found it in connexion with monoplegic paralyses. True, the localization was in one only verified as cortical by post-mortem examination, but on account of what I have said when discussing the cases the diagnosis of a cortical lesion would seem well founded.

By the side of these cases stands, on the one hand, Case XVIII., where a distinct but slight sensory impairment of the fingers passed off during the time of observation, a pure motor paralysis only remaining. On the other hand, we have Cases XVI. and XVII. showing at first no signs of any sensory affection, but where later on disorders of sensibility were added to the motor paralysis, in consequence of fresh lesions; and, lastly, Case XV., in which the sensory disturbance after a fresh lesion extended to a part previously relatively unaffected.

In several cases of cortical monoplegia I found the functions of cutaneous sensibility which I have examined, as well as the muscular sense, to be fully intact in spite even of a highly developed paresis. And since in making these examinations I have been down to the physiological liminal value both as regards cutaneous and muscular sense, this dissociation is at the same time strictly proved.

But the reversed condition may take place in connexion with cortical lesions—viz., that the motility may remain intact or almost intact, whilst the sensibility, and especially the muscular sense, and co-ordination may be affected. Cases of this kind have been clinically investigated by Wilbrand, Lang, Henschen, Jany, Müller, Stauffenberg and others, and in the chapter on localization which follows I shall allude to several such cases where the localization of the lesion was ascertained at the autopsy or during trephining.

To these cases from the literature I can add three of my own—viz., Case XIX. showing disorders, especially of the muscular sense; Case XX. showing disorders of cutaneous and muscular senses; and Case XXI., where on account of remote action an almost isolated affection of the muscular sense appeared, without paresis. In these cases the position of the lesion was ascertained post-mortem. In the former two cases the lesion lay behind the central sulcus, in the last case in the temporal lobe. In none of them was the capsule affected. The same applies to Case XXII., which, however, on account of a fresh lesion, showed later motor disturbances also; here, the localization and extent of the original lesion could not be determined.

From this it follows that in cases of cortical lesions the motility or the sensibility may be highly affected separately, even at an early stage. And my cases go to show that the degree of paralysis present is of no importance with regard to the question whether phenomena of lost function are going to develop within either or both functions.

With regard to capsular lesions it is somewhat different, and to prove this I need only refer to a number of carefully investigated cases related by Henschen, Dejerine, Long and Müller, which all show that, with the exception possibly of very old cases, both motility and sensibility are affected in capsular lesions—a fact which fully agrees with my experience from cases of a typical hemiplegia. Of these it thus holds good that sensibility and motility are simultaneously affected.

I shall not here discuss the anatomical substratum underlying capsular sensory disorders, but content myself with referring to the accounts given by Henschen, Dejerine [47, 50], Long [118], and Müller, which, as far as I can find, do not support the doctrine of *carrefour sensitif* of Charcot.

On the other hand, there is no reason why a cortical lesion, either at the beginning or later, should not affect both sensation and motion, although either may be affected alone. As this cannot occur as a consequence of capsular lesions, we have here an essential difference which is of importance in aiding the topical diagnosis.

Another difference between the sensory disturbances attending cortical and capsular lesions is the extent of loss of function. Then both the motor and sensory tracts have their extensive cortical projection and their narrow passage through the capsule. Just as on the motor side a monoplegia is characteristic for cortical lesions, and a hemiplegia is more often the result of a lesion involving the capsule (although it may attend either of them), so we find in cases of sensory disturbances mono-anæsthesia (hypæsthesia) to be characteristic for cortical lesions, whereas hemianæsthesia (hypæsthesia) appears more often in connexion with capsular lesions.

(a) *The General Distribution of the Sensory Impairment.*

The general distribution of the impairment of sensation is, as a rule, a hypæsthesia increasing distally and laterally, analogous to the distribution of the motor disturbances in ordinary cases. Still, I have elsewhere tried to show that in cortical lesions this distal type of paresis is not universal, cases having been described, although of relatively rare occurrence, in which the paresis has been more pronounced in the more proximal parts of the arm than in the hand; such a paralysis is found in a case related later on (Case XXIII., p. 467).

With regard to sensibility, Schaffer [184] has pointed out that sensation is not always most affected distally, a polyinsular anæsthesia sometimes occurring. Cases of this type have been recorded by Wernicke with distal, transient anæsthesia leaving permanent anæsthetic patches on the upper arm; by Max Löwy with impaired sensibility to touch, temperature, and pain in the middle of the thigh on its external aspect; Gros with anæsthesia of the thumb, index-finger and fossa canina; by Berger anæsthesia of the hand, hypæsthesia of the arm, and of the parts supplied by the trigeminus; by Dercum and Keen, hypæsthesia over the internal surface of the thigh and calf of the leg, with astereognosis of the foot. Besides this, cases have been published by Sträussler and Benedict showing a radicular type of anæsthesia, which they consider to indicate a cortical representation of the spinal segments.[1]

Lastly, it may be pointed out that Oppenheim [158], Pick, Nothnagel [151], and Bramwell mention an anæsthesia following a cerebral lesion which follows fairly closely the distribution of the peripheral nerves; in the two former of a radial type, in the two latter of a median type. These cases, consequently, show that the sensory disturbances need not in every case constantly increase uniformly in a distal direction, at least not as far as cutaneous sensibility is concerned. The impairment of the muscular sense, on the other hand, is, as far as I have been able to gather from the literature, always most pronounced distally; and I have no observation of my own where any disturbance of the one or the other sensory function was found with certainty to be more marked proximally. The condition in Case XVI., where the muscular sense of the wrist-joint was affected although the muscular sense of the thumb and index-finger remained intact, is most likely accounted for by other factors, as will become evident later on.

The rule is, that the impairment of cutaneous sensibility is most marked distally—i.e., in the hand or the foot—but both in the case of motion and sensation it may happen that the disturbances are more marked on the one half of the hand than on the other.

Mills and Weisenburg have related three cases showing impairment of the cutaneous and muscular senses with astereognosis and ataxia of the three ulnar fingers, and one case with transient paresis and paræsthesia of various fingers, and reduced sensibility of the ulnar half of the hand. Similar cases have also been described by Wichura with transient anæsthesia on the radial side of the hand; by Fischer, anæsthesia to touch and pain in the three ulnar fingers and the ulnar side of the hand and forearm, with diminished perception of position and localization in the three ulnar fingers (the author, however, attributes these to hysteria). Kramer described hypæsthesia and atopognosis of the three ulnar fingers, reduction of the muscular sense of the index and middle fingers.

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[1] This theory is adopted by Löwy (on account of two clinical observations); by Muskens (on account of the behaviour of the sensibility in connexion with epileptic fits); by Horsley and Russel (on account of the pre- or post-axial atopognosis). It would, however, go beyond the limits of my investigation to enter upon a critical examination of this question.

In Bonhöffer's [15] case perception of touch and passive movements and position were reduced in the whole of the hand, but most in the three ulnar fingers. Sandberg described reduction of tactile sensation in the whole hand and muscular sense, perception of position and power of localization reduced in the three ulnar fingers. Dana [43] described tactile anæsthesia and analgesia in the forefinger, but no anæsthesia to heat and cold, although there was clumsiness and awkwardness in the fingers and arm; Monakow ([141], p. 671), lasting hypæsthesia and impairment of muscular sense in the three radial fingers; Zenner, hypæsthesia of the arm, muscular sense, and appreciation of position less good in the index and middle fingers; Levi, hemianæsthesia quickly improving, power of localization and muscular sense of the two ulnar fingers reduced; Purves Stewart, muscular sense of the thumb less reduced than that of the other fingers.

In six of my cases the same condition was more or less marked. In Case V., showing a cortico-subcortical lesion within the arm centre, a slight hypæsthesia and hypalgesia was to be found on the radial aspect of the hand. Case XIII. showed a slight degree of hypæsthesia and hypalgesia of the four ulnar fingers; this ulnar hypalgesia, which was very slight, might, however, in this case have been attributed to pure physiological causes—viz., to the less acute sensibility present on the ulnar side of the hand. Case XXII. presented a hypæsthesia of the two ulnar fingers as well as of the corresponding part of the hand; with regard to Cases XV., XVI., and XVIII., the muscular sense only showed this partial and distal form of loss, and this only during one period of the illness. Thus, in the beginning Case XV. presented very marked disturbances with regard to the muscular sense of the four ulnar fingers, the muscular sense of the thumb being somewhat better retained until this was also damaged by a fresh lesion. In Case XVI., on the other hand, no sensory disturbances at all were found at first, but, after a fresh lesion, a marked impairment of the muscular sense of the little finger appeared, and a similar but less pronounced defect in the ring and middle fingers. As regards Case XVIII., at first there was an impairment of the muscular sense affecting all the fingers; later on loss in the little finger only, which ultimately also passed away. Lastly, it is worth mentioning in Case XIV. there was hypæsthesia on the outer but not on the inner aspect of the foot.

In most of the partial anæsthesias of the upper extremity above related, it was either one or the other aspect of the hand that was affected, and when we consider the extensive area of projection belonging to the hand it is almost to be expected that something of this kind should occasionally occur. Still, among the cases just quoted are a few (Kramer's, Dana's, and Zenner's) in which not the one or the other half of the hand was affected, but certain fingers.

A partial and distal hypæsthesia of this kind might, however, have a different significance in different cases. As I have already mentioned, the sense of pain, when tested by the quantitative method (Alrutz's algesimeter), shows a higher liminal intensity on the ulnar than on the radial side of the hand; Mocutchowsky arrived at the same result by his method, which was founded on another principle, and Muskens also came to the same conclusion. Further, the liminal value is higher with regard to the fingers, at least in the terminal phalanges, than in the hand, and higher on the volar than on the dorsal surface. From this it is evident that when estimating slight degrees of hypalgesia in these parts one must be more reserved than in the case of such parts where physiologically no such difference exists. To what extent this factor must be taken into consideration seems to me difficult to decide without a quantitative estimation of the sense of pain. Still, it is obvious that such a slight ulnar hypæsthesia as the one found in my Case XII. is fully explained by the physiological differences between the ulnar and radial side of the hand. The majority of the cases belonging to this group found in the literature are also of this ulnar type, but, on the other hand, partial distal hypæsthesia of the radial half of the hand also occurs, a form which is considered by Monakow to be the more common. When discussing localization I shall return to the significance of these symptoms.

(b) *Dissociated Sensory Disturbances.*

Amongst my cases of cortical lesion, two showed an affection of the muscular sense, but not of the tactile or painful sensations—viz., Case XVI., with motor paresis and disturbance of muscular sense; and Case XVIII., where the muscular sense remained permanently affected although the cutaneous sensory disturbances receded. In the same way Case XXI. showed isolated impairment of the muscular sense in the hand and arm due to remote action. Another case, most likely of cortical nature with a paralysis of very old date, presented a disturbance of the muscular sense without any disturbance of simple cutaneous sensations. This condition was not found in any of those cases of capsular lesion where I was able to make a trustworthy examination of sensation, but it is not unlikely to occur in old cases, as, according to Long and Marie, even the stereognostic loss may show retrogression. In the same way a dissociated sensory disturbance of this kind may occur, especially in connexion with tumours which do not affect the cortex (Monakow, Bullard).

Even if I have not been able to ascertain the absence of cutaneous sensory disturbances in capsular lesions, I have observed cases of hemi- as well as mono-plegia showing minimal disturbances only in cutaneous sensibility, but with very marked disorders of the muscular sense (the cases of hemiplegia I. and V., and the monoplegias XIV., XV., XVII., and XX).

On the other hand, in no case of either cortical or capsular lesions could I find an affection of tactile sensation without a simultaneous marked disorder of the muscular sense. According to the literature this seems to be the rule, although this statement is opposed by Gordon [74], Henschen, and, to a certain extent, by Oppenheim. The opinion expressed by Henschen and Oppenheim, which differs from the prevalent one, may possibly be explained by the fact that these authors in their investigations have paid special attention to sensations of position and have habitually tested them by letting the patient imitate with the non-paretic hand passive movements of the paretic one, a method which, according to my experience, is not sufficient to exclude completely the presence of disturbances in the appreciation of position.

From the above it is evident that important differences between cortical and capsular lesions are to be found with regard to the frequency in the occurrence of sensory disturbances and with regard to their distribution: in connexion with cortical lesions we often find purely motor, or even purely sensory, paralyses; in connexion with capsular lesions the paralysis is, as a rule, sensori-motor. After cortical lesions mono-anæsthesia is more often found, after capsular lesions hemianæsthesia. Lastly, a dissociated sensory impairment more often occurs in the case of cortical lesions than in those of the capsule, the muscular sense in that case being affected alone.

As to the form of disturbances present—i.e., the differences in quality in connexion with lesions in one or other situation—there exists, according to my experience, purely a gradual difference, the disturbances of cortical origin, as a rule, not being so intense as those due to capsular lesions. Nor have I found in the literature mention of any other difference than one of degree.

In this connexion it must, however, be remembered that certain functions, which lie for the most part beyond the scope of this work, require at least partially the presence of the motor-power if they are to be more closely observed. On this account the observations made on this point are not, without further investigation, transferable to hemiplegias of capsular origin, accompanied by disturbances of the so-called sense of strength, apraxia, &c.

(c) *The Power of Localization.*

During my investigations I have tried to arrive at a comparison between the simple tactile sense and its associated function of a higher quality—the power of localization. Under normal conditions the power of localization is developed to a different degree in various parts, and is in no way proportional to the acuteness of tactile sensation in that part of the body. In the upper extremity the power of localization grows keener in a distal direction—an important fact, considering that this part of the body is most often affected in the cases described in this paper.

Consequently, there is nothing surprising in the fact that the disturbances in the power of localization following a cerebral paralysis are not always more pronounced distally. Thus, Sandberg and Hatschek found the power of localization affected on the trunk only, while others—Bonhöffer [15], Horsley [90], Cadwalader, Mills and Frazier, Oppenheim [158], and Schittenhelm—observed disturbances chiefly distally. And in all my cases, capsular as well as cortical, the impairment in the power of localization was most marked distally (except in Case XXII.); the disturbances of the hand and forearm were often very great in spite of the fact that there were no signs of impairment proximally.

With regard to the relation between disturbance in the power of localization and disorders of the simple "tactile sensation," different opinions exist. Müller and Schittenhelm are of the opinion that profound disturbance in the power of localization only occurs in connexion with disorders of tactile sensation; whereas, on the other hand, Wernicke, Hoppe, Levi, Bruns, Walton and Paul [217], Purves Stewart and Monakow [141] relate cases showing impairment in the power of localization without simultaneous disturbances of cutaneous sensibility. Förster [67] points out that the atopognosis found in tabes and central lesions as well as in peripheral neuritis, and even in affections of the joints, is connected with disturbances of the so-called "articular sensibility," from which he draws the conclusion that topognosis depends upon the sensations of position, the former disappearing when the extremity is too little used. Too great importance should not be attached to this parallelism, or at any rate it is not without exceptions, as is shown by Förster's own Case 19, and still more plainly by a case related by Sträussler, where the patient could record the direction of the movement correctly, but not state which finger was being touched. Normal topognosis in connexion with disorders of the sense of position is also mentioned by Starr and McBurney, Oppenheim [155], and Schittenhelm. Sandberg believes he has found a connexion between topognostic disturbances and disorders of deep sensibility. Any connexion of this kind I was unable to prove, as I, like Müller, never noticed an affection of the deep sense of pressure without a simultaneous affection of tactile sensation.

From the above it may be gathered that topognostic disturbances may occur in connexion with disorders of sometimes the one and sometimes the other function, but may also occur quite independently. In my cases an impairment of the power of localization was never found without simultaneous disturbance of the muscular sense. Impairment of the power of localization in spite of intact tactile sensation was shown in Case XVI. But I could never prove the reverse condition, nor was this, as a matter of fact, to be expected.

In many cases the disturbance in the power of localization follows undoubtedly an irregular type. For instance, in a case related by Schaffer [184], where the lesion was of capsular origin, the patient localized a touch on the lip to the thigh, &c. Similar cases have been observed by Oppenheim [158], and in many cases a considerable and perfectly irregular atopognosis occurs, even if not of such marked degree. In some cases, however, and possibly in the majority, a certain regularity with regard to the atopognosis is to be found. Müller, for instance, has found that the patient localized the touch to the respective extremity as a whole. Verger [219] found that the patient usually localized too far proximally "in a direction towards better sensation," as Verger puts it, and, according to Horsley and Russel, this type of atopognosis would seem to be the one occurring most regularly. This rule is, however, not without exceptions, as appears from two cases by Horsley and Russel on the one hand, and from one case by Morton Prince on the other.

Besides this, Horsley and Russel have drawn attention to another form of atopognosis, which may occur simultaneously with the other. They describe an atopognosis towards or away from the mesial axis of the arm; in their cases it has only been a question of the upper extremity. This atopognosis follows a direction at right angles to the proximal atopognosis previously mentioned.

I did not have an opportunity of observing a perfectly irregular atopognosis, except in some cases of old hemiplegia, as well as in those cases where the attention of the patient could not be sufficiently attracted.

In those cases, on the other hand, where a certain degree of regularity was present, this was of a proximal type, although varying considerably in degree (III., IV., XIII., XIV.), and in two cases, Nos. V. and XVI., a transverse atopognosis—viz., in the former case a post-axial and in the latter a pre-axial atopognosis, besides which the former case also showed a proximal atopognosis.

As previously mentioned, Verger has pointed out with regard to the proximal atopognosis that this takes place in a direction towards better sensation. This condition I also found in those two cases of transverse atopognosis which I have had an opportunity to observe.

(*d*) *The Muscular Sense.*

I have paid special attention to the muscular sense, or, more correctly, one function of it — the perception of passive movements — and my intention was to make a more detailed estimation of the quantitative disturbances in different joints and in connexion with different directions of movement. I also tried to investigate whether, with regard to disorders of this function, any difference could be ascertained between capsular lesions on the one hand and cortical on the other. I was, however, unable to discover any other difference except that in the former case changes in the muscular sense were found to be constant, but often failed completely in the cortical cases.

Here I must first lay stress upon one point to which, it is true, previous attention has been drawn (Wernicke, Bourdicaud-Dumay, Bonhöffer [15] and others)—viz., that with regard to the muscular sense a definite difference exists in many cases between the perception of a movement, *per se*, and the appreciation of its direction, the latter function alone being at times affected. The reversed condition, naturally enough, never takes place. Just as in the case of tactile sensation a distinct difference exists between a simple and a more complicated function, viz.—the difference between appreciation of a touch as such and the appreciation of tactile localization—in the same way with regard to the muscular sense, a difference exists between perception of a movement and perception of its direction. This difference—viz., a disturbance much more marked with regard to the more complicated function represensed by perception of direction than with regard to the simple function, appreciation of movement pure and simple—is to be found in several of my cases, the cortical as well as the capsular ones (I., IV., V., XIII., XIV.).

As no numerical estimates with regard to these disturbances, as far as I can gather, have been published, it seems to me appropriate to collect in a tabular form the results to which I have come when trying to define the minimum movement perceptible in different parts of the upper extremity, although these results have been obtained by the aid of an approximative method only.

As will be seen from this table, the disturbances of the muscular sense were always more marked distally in these cases; and the same condition evidently prevailed in those cases where no such quantitative estimation had been made. Thus, at seven examinations of four cases it was observed that the muscular sense was completely absent in the joints of the hand and fingers, although no affection of the shoulder-joint could be discovered. On the other hand, in two cases only was I able to ascertain any—and even then only minimal—perception of movements in the joints of the hand and fingers, the impairment of the shoulder-joint being at the same time evident. When, however, the different results of the investigations are compared, one finds that this reduction, which increases distally, does not show any uniformity with regard to the different joints in different cases, or even at different examinations. This is most obvious when comparing V. and XV. On this account it is not inconceivable that cases might occur in which the muscular sense would be more affected proximally than distally. I have, however, not been able to find any quite certain cases of this kind.

I wish to point out the following fact with regard to disturbances of the muscular sense. Movement of a certain joint is not uncommonly appreciated better in the one direction than in the other. Mills and Weisenburg found the same condition in one case (Case 2); when the relaxation was almost perfect, it seemed as if the loss of the sense of position was more marked when the fingers were bent backwards—that is, extended—than when they were bent forward to the palmar aspect of the hand. This condition, which I have found mentioned by Mills and Weisenburg only, I observed in Cases I. and XVI. In Case I. extension at the elbow was better appreciated than flexion. In Case XVI. it was the wrist, and here movements of flexion were better perceived than those of extension.

CASE	SHOULDER-JOINT		ELBOW-JOINT		WRIST-JOINT		FINGER-JOINTS	
	Movement	Direction	Movement	Direction	Movement	Direction	Movement	Direction
I	min.	5°	extension min. flexion 10°-15°	10°-15° 15°-25°	max.	not even max.	max.	not even max.
III	—	min.	—	5°-10°	30°	—	not even max.	—
IV	—	min.	min.	> 15°	45°	not even max.	not even max.	—
V	5°	15°	10°-15°	45°	max.	max.	little and ring finger max. other fingers not even max.	—
XIII a	45°	90°	not even max.	—	not even max.	—	not even max.	—
,, b	—	45°	not even max.	—	not even max.	—	not even max.	—
,, c, d	min.	min.	—	20°	not even max.	—	not even max.	—
XIV	min.	5°	min.	> 15°	—	not even max.	> min.	not even max.
XV a	—	min.	—	min.	> min.	30°	thumb other fingers 45°	30°
,, b	—	min.	—	min.	min.	min.	thumb min. other fingers	min. 30°
,, c	—	min.	—	min.	> 15°	—	all fingers not even max.	max.
[1]XXIVa	min.	1°-3°	2°-3°	10°-12°	30°	45°	30°	45°
,, b	min.	min.	min.	> 2°-4°	min.	10°	min.	10°

In this table the first column contains for each joint the angles at which movement pure and simple is perceived; in the second column the angle at which the patient appreciated the direction of the movement. (Max. means maximal; min., minimal movement; a, b, c, different examinations.)

[1] This case is not reported in full in the text.

The physiological substratum underlying perception of passive movements is differently given in the various physiological text-books. One opinion which is very commonly expressed is that the articular sensibility forms the foundation for this perception. But the articular cartilages can hardly be considered as the basis of these sensations, since, as proved by Lennander, they are devoid of sensation. The capsule is evidently that part of the articulation which must be considered as the source of this function. Besides this, the stretching of the skin and soft parts plays a part. Cases which show a different perception of the movements of the same joint with movements in different directions seem to me not quite unlikely to contribute to the solution of this question. Firstly, it is evident that the articular cartilages cannot be the source of this sensibility, even if Lennander's investigations are entirely left out of consideration; for it is the same articular segments that come into contact in the one movement as in the other, although the movement takes place in different directions. Passive stretching of the capsule and the skin is also likely to have the same effect in both forms of movement. With regard to the muscles, on the other hand, the condition is somewhat different. In Case I. the flexor muscles possessed a greater tone than did the extensors, and consequently the movement of extension stretched muscles more powerfully contracted than was the case with flexion; the former movement would therefore have a greater chance of making itself perceptible than the latter. Case XVI., on the other hand, presented no contracture, but for several months the patient, while using a sling, had been in the habit of letting the hand hang down in the volar flexed position. In consequence it was the movement which acted in the same direction as the force of gravity, which more easily escaped the attention of the patient.

Further, in a case of amyotrophic lateral sclerosis, I have found what seems to me a very similar condition. Here the elbow was in a condition of flexion-contracture and minor movements of flexion easily escaped the attention of the patient; but when the patient was particularly asked to pay careful attention to the matter, she observed minimal movements of flexion with the same degree of certainty as movements of extension. Thus it cannot have been the tone of the muscles solely which played a part in this case. It is conceivable that movements in the same direction as the involuntary tonic contraction (Case I.), or in the same direction as the force of gravity (as in Case XVI.), might more easily escape attention than a movement in the opposite direction. A similar explanation might be given for the disturbance of muscular sense on volar flexion of the wrist presented by Case XVI., in spite of the absence of such impairment in more distal joints, such as those of the index-finger and thumb; although no typical passive contracture of the flexors of the forearm had yet taken place, the tone of these muscles was greater than that of the extensors of the forearm, which were constantly being stretched.

With regard to the much-debated question concerning the peripheral substratum underlying the muscular sense, these observations seem to me not without interest; although they are much too scanty to allow of any conclusion; still, it seems to me they go to show that not only the articular sensibility and cutaneous sensations, but also the muscles play a part in the perception of passive movements.

(e) *Cortical Ataxia.*

Of all the sensory disorders tested, disturbance in the perception of passive movements was not only that most frequently met with, but was also most permanent. There were, however, several cases in which no such disturbance was discovered even a few days after the onset of a severe motor paralysis, showing that motor disturbances and impairment of the muscular sense may occur independently of each other.

This is of importance for the knowledge of cortical ataxia. This does not consist of an abnormal movement, which has added itself to the voluntary movement, as is the case in chorea, athetosis, &c., but, like the ataxia of tabes, is due to a disturbance of the voluntary movement itself, which is altered in direction and precision. Thus we have to deal with a real inco-ordination which, however, does not by a long way reach the same degree as in tabes (Dejerine [48]), although it is often combined with very marked sensory disturbances, which may completely extinguish the muscular sense, the power of localization, the sensation of position, and other more complicated functions.

Further, it is especially the more isolated and acquired movements of a complicated nature which are damaged while the coarse power may be fairly well retained (Monakow [141]). Thus cortical ataxia approaches apraxia, by which is meant inability to perform certain specially acquired movements in spite of well-retained motility (Liepmann [113, 114, 115]). In the majority of cases it would, according to Wilson [229], seem possible to differentiate between cortical ataxia and apraxia. The cortical ataxia becomes evident in all the movements of the extremity in question; in the case of apraxia, on the other hand, certain movements can be perormed without any trace of ataxia. Further, the degree of cortical ataxia is different according to the different extent of the movements: the smaller and simpler the movement, the less evident the ataxia; the greater and more complicated the movement, the greater the ataxia. The apraxia, on the other hand, shows no such regular connexion between the magnitude of the affection and the degree of complexity of the movement. The errors which occurred gave more the impression that the direction of the movement or the way to complete it had been forgotten, the movement being from the beginning wrongly innervated.

I have not investigated these complicated disturbances, but have restricted myself to the more simple functions and disorders. Still, I have found that not only the coarse power but also isolated finger-movements (Case XX.), and even certain complicated acquired movements (Case XXI.), may be well retained at the same time that impairment of the muscular sense is obvious. In Case XX., however, disturbances of associated finger-movements appeared later, as far as could be made out, on account of the bad condition of the patient. Case XXI., as well as a case recently published by Stauffenberg, show that a function as complicated as the fastening of a button, &c., may be retained in spite of sensory disorders and in spite of disturbances in the muscular sense and the orientation in space. Thus impairments of the muscular sense need not be attended by a complete cortical ataxia. On the other hand, we find, as Monakow points out, that cortical ataxia is not always combined with impairments of the muscular and cutaneous senses, although this is very often the case. This coincides with the fact that a more complicated function, in this case the co-ordination, may be affected although one of its components (perception of movement), or even several of its constituent parts, may be unimpaired; whereas, on the other hand, there is no support for assuming the reversed condition to take place. Besides it is, as a rule, especially tumours (Dejerine [48]), which give rise to a higher degree of cortical ataxia, and not only those situated in the sensory-motor area (Bruns [26]), but even tumours situated elsewhere—for instance, in the cerebellum, as I myself have had an opportunity to observe in one case.

(*f*) *Orientation in Space.*

One of the functions which takes part in the building up of accurately balanced and well co-ordinated movements is the orientation in space of any particular extremity at rest as well as in motion. This function, to which my attention has been drawn by Horsley and Slinger's interesting work, I have examined in seven cases (Cases IX., X., XI., XVIII., XXI., and two others). Of these cases, IX., X., and XI. were motor monoplegias purely; Case XVIII. was a sensory motor monoplegia in which the sensory disturbances finally disappeared entirely; Case XXI., presented a disturbance of the muscular sense without any paresis. In Cases IX., XI., XVIII. and XXI. the paresis was not so marked that the patient could not move the paretic hand towards the non-paretic one. In Case X., on the other hand, the paresis was of such a degree that the examination could only be made by approximating the non-paretic hand towards the paretic one. In the rest of the cases either the paresis or the hypæsthesia was of such a degree as to render the method useless.

Now it became apparent, as was to be expected, that in Case XXI., which presented marked disturbance of the muscular sense, the orientation in space was considerably affected. In another case showing only minimal disturbances of the muscular sense, the impairment with regard to orientation in space was uncertain. On the other hand, Cases IX. and XI., in spite of the absence of any disturbances of the muscular sense, showed alterations with regard to orientation in space. No disorder of this function was found in Case XVIII., and the same applies to the case of amyotrophic lateral sclerosis previously cited.

That the muscular sense—on the whole one of the most important functions with regard to exact co-ordination (Leyden)—also plays a part with regard to orientation in space is evident, and thus the relation between the disturbances of the muscular sense and orientation in space becomes analogous to the relation between disturbances of the muscular sense and astereognosis. For, as pointed by Dejerine [48, 51], disorders of a higher function are the necessary correlative of disorders of the simpler function which forms an integral part of it. This is the reason why there is nothing astonishing in the fact that cases showing disorders of muscular sensation also present disturbances of orientation in space. It is of greater interest that orientation in space may be damaged without the muscular sense presenting any alteration (Cases IX. and XI., and perhaps Case X.).

Co-ordination, including orientation in space, presupposes the conjoint action of several simpler functions, and evidently also of motor centres. Consequently a disturbance in different directions might each one by itself lead to inco-ordination. From my Cases IX. and XI. it is evident that motor disturbances may give rise to disorders with regard to orientation in space without the perception of passive movements being at the same time damaged. This agrees with Monakow's experience that disturbances of the muscular sense are not always present in cortical ataxia. Lastly, it is of interest to see that sensory disturbances attending a cortical lesion which at first were present to a high degree may disappear so completely that not only the muscular sense but also the orientation in space are completely restored (Case XVIII.). Case XVIII. and that of amyotrophic lateral sclerosis show that such disorders of orientation in space do not always attend lesions of the pyramidal tract.

Further, when testing the orientation in space I found the condition described by Dejerine [46] with regard to sensations of position—viz., that these are better when the arm is in motion than when at rest. This condition appeared in Cases IX. and XI. In these the error became less marked when the paretic arm was moved towards the non-paretic one than *vice versa*, in spite of the injurious effect of the paresis, and in spite of the intentional tremor presented by Case IX. being most marked in the paretic arm. In Cases X. and XXIV. this examination could not be carried out on account of the considerable degree of paresis present. The case in which I was able to show that no such difference existed (Case XXI.) did not present any paresis, but only an affection of the muscular sense.

The difference between the orientation at rest and in motion became still more evident on comparing the results obtained in Case XI., by Horsley and Slinger's method, with those obtained by Blix's method. The former method revealed errors far in excess of the normal; the latter showed that the orientation of active movement took place almost equally well with the paretic arm as with the non-paretic one. Unfortunately this method, which requires fairly good motor function, at least in the shoulder- and elbow-joints, could not be employed except in this case and in Case XVIII., both of which showed normal conditions.

The error in the perception of the position of the hand is normally more often of such a kind that the hand is estimated to be in a higher plane than that in which it really lies. This condition Horsley and Slinger found to be the rule with regard to the overhead position; here this mode of error was characteristic. In those of my cases which presented a paresis in connexion with a disturbance in the orientation in space, this direction with regard to the faulty perception was constant, the position of the paretic hand being estimated higher than what it in reality was, as has been observed by Westphal. In my cases it appeared, furthermore, that the error increased in those planes which were situated higher up, as is the case under normal conditions, but this took place to a proportionately greater degree than normal. Case XXI., which showed disturbances of the muscular sense but no paresis, formed an exception to the rule that the position of the hand was estimated too high.

From this it appears that motor disturbances may give rise to disorders of orientation in space, although not constantly. Whether the motor disturbance by itself can in any way influence the form of disturbance found in connexion with this function I must leave undecided, my material being too limited to allow of any conclusions on this point.

CHAPTER V.—THE CORTICAL LOCALIZATION OF SENSATION.

§ 1.—*Recent Anatomical and Physiological Investigations upon the Cortical Localization of Motion and Sensation.*

THE problem of the cortical localization of sensation is intimately connected with the problem of motor localization.

Hammarberg, Ramon y Cajal, Brodmann, and Campbell have found considerable histological differences between the anterior and posterior central convolutions. In the anterior central convolution the cortex layer is much thicker than that of the posterior (Brodmann). In all the layers of the cortex of the anterior convolution (Campbell) a greater abundance of radiating nerve fibres are found than in any other part of the cortex. Within the same area, and, practically speaking, limited to it, though not occupying its entire extent, are situated the giant-cells of Betz (Campbell's "precentral area"). In the posterior central convolution the nerve filaments are far less numerous than in the anterior, but, on the other hand, inter-radiating plexuses of coarse calibre cross the radiating bundles (Campbell). In the posterior central convolution the giant-cells of Betz are absent. The numerous stellate cells, on the other hand, form a definite layer in this part of the cortex. According to Hammarberg, however, there are, in the superior sixth no greater cytologic differences between both gyri centrales.

Campbell, further, considers that he is able to distinguish two different zones in the posterior central convolution by means of their histological structure. In the anterior half of the gyrus ("post-central area proper") there are numerous large pyramidal cells, as well as the inter-radiating plexus of large calibre. In the posterior half of the convolution, which forms a transition to the type found in the temporal and parietal convolutions, the large pyramidal cells and fibres of large calibre disappear. The chief difference between the cortex covering the posterior part of the central gyrus and that covering the gyri parietales, according to Campbell, consists in the layer of Baillarger, which in the latter area is easily seen. Brodmann also divides the gyrus centralis posterior, on account of its histological structure, into different territories, but differentiates three such areas.

According to Flechsig and Vogt, a difference exists between the myelinization of the gyrus centralis posterior on the one hand, and that of the gyrus centralis anterior, together with the parietal region, on the other; the myelinization of the gyrus centralis posterior beginning earlier than in any other part of the cortex.

From the anterior central angle proceeds the pyramidal tract, as proved by the investigations on amyotrophic lateral sclerosis and on the *réaction à distance* following lesions within the motor path. To the posterior central convolution pass, on the other hand, the cortical tracts of the optic thalamus, as has been shown by Roussy. In one case of thalamic lesion he was able to follow, by Marchi's method, the degenerated tract to the posterior central convolution. The topography of the thalamo-cortical neuron may, according to Hösel, be well observed in a foetus of nine months, where this path, but not the pyramidal tract, has acquired its medullary sheath. It is situated in the posterior part of the internal capsule, or, more accurately, in the second and third of the five capsular segments. It runs more in a direction backwards than the pyramidal tract, and, in consequence, a partial crossing of these two paths takes place, as pointed out by Fr. Müller.

Experimental physiology has added some important contributions to our knowledge of the motor projection. I shall, in this connexion, altogether neglect the earlier literature on this subject, which may be found in the ample accounts by Soury and Monakow [140, 141], only drawing attention to Sherrington and Grünbaum's experiments on the higher monkeys (gorilla, orang-outang, chimpanzee). By means of unipolar excitation they were able to verify the results obtained by Hitzig, to the effect that, broadly speaking, the excitable area of a limb was localized to the anterior, and did not extend on to the posterior, central convolution. Besides this, they found that the position of the different centres varied considerably in different individuals, remembering that the centres can only be identified with full certainty by means of excitation. Further, it became apparent that the sulci of the brain were of approximate importance only, in marking out the boundaries of the physiological areas of projection. Thus, for instance, in some individuals contractions could be evoked from the posterior wall of the sulcus centralis, in others this part was inexcitable. They also found that cortical stimulation behind the sulcus centralis, which, by itself, could not provoke contraction, facilitated or increased the effect of a stimulation in the gyrus centralis anterior, if this was applied in the same horizontal plane.

Rothmann, from experiments on macacus and rhesus, is not in complete agreement with the view held by Sherrington and Grunbaum; still, he admits that the anterior convolution, more especially in the region of the arm, is much more easily excitable than the posterior one.

As is well known, Horsley and others have described the excitable area in man as extending to both central convolutions. Krause, on the other hand, considers the anterior convolution only to be excitable, and Mills and Frazier [134] show that excitation much more easily produces an effect from the anterior central convolution; but when a stronger current is used, or the anterior convolution has previously been excited, then the posterior central convolution also becomes excitable.

Lastly, in certain pathological conditions the histological examination has shown important differences between the anterior and posterior part of the gyrus centralis posterior; and we are entitled to draw conclusions regarding the function belonging to these parts—viz., in amyotrophic lateral sclerosis as regards the motor, and in thalamic lesions and tabes as regards the sensory functions. Campbell found, after amputations and in a case of capsular hemiplegia, that the *réaction à distance* was most pronounced in the gyrus centralis anterior; a distinct if not highly marked, alteration also occurred in the anterior half of the gyrus centralis posterior.

In the case of a lesion within the brain itself different tracts might, however, have been affected, and on this account Campbell's result as regards a capsular lesion is not incontestable. Holmes and Page May avoided this source of error by studying the cortical changes after hemisection of the cervical region of the spinal cord in dogs, cats, lemurs, monkeys of the macaque family, and in a chimpanzee; they also investigated two cases of acute traumatic transverse lesion at the seventh cervical segment in man. The pyramidal tract being the only direct cortico-spinal path, it follows that those cortical cells which showed *réaction à distance* after a lesion affecting the medullary pyramidal tract must constitute its place of origin. By this means they proved, at least with regard to the extremities and trunk on the one hand, that the origin of the pyramidal tract is situated in front of sulcus centralis contralateralis, on the other that the pyramidal tract issues from the cells of Betz only; for these cells are the only ones showing *réaction à distance.* They found, it is true, that after hemisection on monkeys about one-fifth of the whole number of Betz cells did not undergo degeneration, but by means of electrical stimulation they showed that these cells belonged to the pyramidal tract of the same side.

In full agreement with Holmes and Page May's investigation stands the curious case of intracranial hemiplegia related by Spilmeyer; here there was within the cortex an intense destruction which had left untouched one kind only of the specific elements—namely, the cells of Betz. In this case the pyramidal tract was fully intact.

The functional difference between the two central convolutions is not less evident from the histological appearances found in amyotrophic lateral sclerosis. This disease constitutes, as is well known, a degeneration localized to the motor neuron-chain, a degeneration which may be followed to the cortex. In a case of this disease with a quick course, and showing early and prominent bulbar symptoms, Lennmalm found a rich collection of *corps granuleux* in the medullary substance of the lower part of the central convolution. Otherwise they were sparingly and more irregularly scattered within both central convolutions. Spiller [190], in one case of the same disease, found by the aid of Marchi's method degenerated fibres in the posterior as well as in the anterior central convolution, but considerably more in the latter. Still more instructive are the cytological investigations made in cases of amyotrophic lateral sclerosis, especially by Campbell, Rossi and Roussy. The first-named author found in two cases that the changes were localized strictly to the gyrus centralis anterior, and that they had affected especially the giant-cells of Betz, the cells characteristic for this convolution, as well as the radiating bundles of fibres. Rossi and Roussy found in three cases the same changes in the anterior central convolution, but they were also able to demonstrate in one case a slight extension on to the posterior central convolution.

Thus the distribution of the cortical changes in amyotrophic lateral sclerosis indicates with certainty that the anterior central convolution possesses a motor function, and that such function cannot be attributed to the gyrus centralis posterior in anything like the same degree.

These investigations are amplified by Roussy's observations in connexion with some other processes. In one case of lesion of the optic thalamus Roussy, by means of Marchi's method, could follow the degenerated thalamo-cortical fibres, which must be looked upon as a sensory tract right up to the central convolutions, and was able to demonstrate that the majority of the fibres passed to the gyrus centralis posterior.

Lastly, in connexion with parietal lesions, Monakow [141] was able to show a centripetal degeneration of the lemniscus.

In this short review I have only tried to set out the more important results obtained during the last few years. From these it is obvious that considerable differences exist with regard to the construction of the two convolutions; further, as the result partly of experimental, partly of clinical evidence, the anterior central convolution must be regarded as having a motor function of which the posterior central convolution is devoid.

With regard to the posterior convolution, it is probable (p. 448) that its anterior half stands in nearer relation to the peripheral organs than does the posterior half, and it seems obvious that at least some sensory functions are represented in the posterior convolution.

The experimental method of investigation has, for obvious reasons, not given equally good results in the case of the sensory as in the case of motor functions. This applies more especially to experimental excitation, which, of course, has been applicable to man only. As to experiments on man with a view of ascertaining the sensory function by means of excitation, I have found in the literature only very scanty statements of somewhat older date. Nor are the results incontestable, as it is not possible to estimate the part which a simultaneous action on the vaso-motors might have played. According to Oppenheim [158], the connexion between vaso-motor and sensory disturbances is very intimate. On the other hand, after an incision into the motor area the same authority has observed vaso-motor disturbances in the contra-lateral side, from which it follows that it is impossible to decide whether the sensory effect of the excitation has been primary or secondary. On this account I shall only briefly refer to these experiments. Making use of a defect in the skull after trephining over the central region, the investigators have been able to excite the brain by means of electrodes without the use of an anæsthetic. The results obtained by Bartholow, Ransom, Dana [42], and Laycock, Negro and Oliva, showed that the patient developed paræsthesiæ of rather varied character in the different cases, it is true, but constantly localized to the contra-lateral side. The three former found a simultaneous sensory and motor effect with the same localization. In Negro and Oliva's (cit. Brécy) experiments a sensory effect was obtained with a weaker stimulation than was necessary to produce a motor effect.

Laycock was able to produce sensory excitation without simultaneous motor effect. When the stimulus was applied to the brain in the lowermost part of the osseous defect, sensations in the neck were felt; when the application was made higher up, sensations in the arm and down the hand were produced; and, lastly, an application in the upper part resulted in sensations being felt in the shoulder. This experiment indicates that the sensory projection is divided into different territories for different parts of the body, in the same way as the motor projection. The sensations were not painful, but of a "tingling" character. The results were more decided in the case of cathodal than anodal excitation.

Ransom mentions briefly that the muscular sense improved when the muscles contracted after stimulation, and that this effect lasted for a few minutes after the application of the current.

In Ransom's and Laycock's, and probably also in Dana's, case the stimulus was applied to the anterior central convolution. With regard to Bartholow's case, no information is given on this point.

The theoretical interpretation of these experiments is still a subject of discussion, especially when a case, like the one related by Laycock, goes against the experience gained later on. At any rate, one cannot, as Brécy has done, draw the conclusion from them that they prove an identical projection of motion and sensation. Even if we neglect the part which might have been played in these experiments by the vaso-motors, the chance of a diffusion of the stimulus through the arachnoid fluid was much too great; for the conductive resistance of the fluid forms only a small fractional part of the conductive resistance of the nervous substance itself (Göthlin).

§ 2.—*Clinical Investigations.*

(*a*) *Common Sensory-motor Area or not?*

The experimental investigations with regard to the localization of sensibility performed on animals have had reference to the symptoms of loss function only; but results obtained through experiments on animals, however well carried out, are not directly applicable to man, more especially when the respective organs present such great differences as do the human brain and the brain of the animals in question. For this reason, while admitting the great importance of these experiments for the development of the doctrine of localization, I shall not here deal with them fully, and shall refer the reader to the accounts on this subject given by Soury and Monakow [140, 141].

The most important foundation for our knowledge with regard to the sensory localization in the human cortex is the clinical observations on sensory defects consequent upon cortical lesions. Exner, Dana [43], and Lisso have shown, by means of extensive lists of cases collected from the literature, that in the majority of cases of lesions within the central convolutions sensory and motor disturbances occur simultaneously; since then clinical and anatomical investigations have added numerous contributions that do not permit of any doubt as to the possibility of a cortical sensory disturbance. Before passing to a final review of these clinical cases gathered from the literature, I shall mention a few facts of importance when judging a heterogeneous set of cases of this kind collected from different authors. First, it is clear that such cases that have been a subject of anatomical, and also ultimately histological, examination are those that give the most certain material for judging the locality and extent of the lesion. The certainty is naturally less with regard to lesions verified only by trephining on account of the total view of the case not being so good, and the possibility that multiple lesions may be overlooked. As regards injuries, the locality of the lesion is often pretty well defined by the place of the trauma, but it is difficult or impossible to recognize the extent of the lesion. Lastly, with regard to the cases that have only undergone a clinical examination, it is, as I have previously pointed out, often possible to decide whether the lesion was cortical or capsular, but it is difficult to determine the area and extent within the cortex which was occupied by the lesion.

Different kinds of lesions also have different value with regard to localization. Tumours and abscesses are in this respect worst of all on account of the diffuse changes which may be found outside the area of the lesion itself. These may make themselves noticed histologically (Redlich [170]), or may arise from processes of a functional nature—a diaschisis. This, on the other hand, explains the mixture of local and remote action which is characteristic of tumours, and which may highly complicate the picture presented by the disease. Further, the symptoms of lost function do not always appear with the same regularity in connexion with these processes as in the case of those vascular lesions with an acute onset, and they may sometimes be altogether missing, as in two cases of tumour in the motor area without paralysis related by Byrom Bramwell [17]. Whether the axis-cylinders, proved by Marburg to be present inside tumours, can in such cases help in maintaining function is, after all, uncertain when we consider the distinct pathological changes presented by the axis-cylinders. In spite of these objections a large number of investigations dealing with this subject made during recent years are based on cases of tumour or abscess.

In my collection I have included almost entirely cases where the lesion was situated above the internal capsule. Still, it must be pointed out that the cases I have quoted do not altogether deserve the name of cortical ones. This applies especially to cases of thrombosis, the most common local cortical lesion of vascular nature. For the branches which enter the brain to go to the surface are of two kinds, partly purely cortical and partly medullary. When injecting an area of arterial distribution, its purely cortical branches are first filled and afterwards the medullary ones—i.e., a greater pressure is needed in order to fill the latter (Monakow [141]). On this account the consequences of an ischæmia are first noticeable in the arteries of the medulla, and later on in the cortex. The expression "cortical" is thus somewhat incorrect when speaking of a thrombosis. Similarly in the great majority of cases of local lesions of some other nature —as, for instance, in tumours—the process has had time at autopsy, during life or *post mortem*, to affect the medullary substance of the brain, even in cases where the lesion has started from the cortex. Traumatic lesions or affections starting from the membranes of the brain may, however, produce a purely cortical destruction.

Although the expression "cortical" is undoubtedly incorrect in the majority of cases, I have still used it in the following connexion in contradistinction to capsular. The cases I have collected have been those where definite and, in the majority of cases, positive statements are to be found with regard to sensory disturbances in connexion with cortical lesions. It would have been a great advantage if, in addition, I had been able to relate those cases from the literature where a definite negative statement is to be found concerning disorders of sensation. Slight sensory disturbances, however, are often difficult to detect, and may thus have escaped attention in many cases which have not been specially examined with regard to this point. A short statement of this kind—"no sensory disturbances"—is in such cases of very little value.

The question of cortical localization of sensation is intimately connected with the question whether a common sensory-motor zone exists or if there are different areas of projection for the motor and sensory functions. A chiefly sensory-motor zone is accepted by several investigators—Dana, Henschen, Dejerine, Long, Verger, Horsley, and others—and the region of this projection is said to consist chiefly of the central convolutions. Henschen, however, when discussing one of his cases ([85] Case 16]), asserts that the sensory function is more represented in the posterior central convolution. The localization of a common sensory-motor zone in the central gyri was made before the appearance of the facts that point with such decidedness to a limitation of the motility to the anterior central convolution, and the authorities above mentioned have not since then reconsidered their position with regard to the question, as far as I have been able to make out. Long [119], it is true, accepts the supposition that motility is limited to the anterior central convolution, but, at the same time, he does not mention anything about his position with regard to the above question.

In the previous part on the symptomatology of sensory disturbances I have pointed out that cases exist where the sensory or the motor function may be separately affected as the result of a cortical lesion, and it is consequently, possible to meet with cases showing purely motor or purely sensory loss of function. This may happen at a time when there can be no question of a substitution on the part of other centres and tracts. This proves that sensation and motility have a different localization in the cortex, a view which is accepted by Mills, Spiller, Oppenheim, Monakow, and others.

It next remains to consider the topographic position and the extent of the sensory projection on the surface of the hemisphere. The frontal lobe may be at once excluded, and the fact that sensibility cannot be localized to the anterior central gyrus will necessitate its localization behind the sulcus centralis. This is in agreement with Roussy's case of thalamic lesion, where the degenerated fibres were found to go chiefly to the posterior central convolution. Further, it agrees with Campbell's conclusions, and, lastly, it is supported by the results of the more recent clinical investigations.

As a matter of fact, there are a considerable number of cases to be found in the literature where the sensory disturbances have more or less predominated, or where disturbances of motion have been almost entirely absent or extremely slight, and the lesion was situated behind the sulcus centralis. Of this condition instances are to be found in the cases by Binswanger (Case 3), Broadbent, Bruns (Case 2), Burr, Cadwalader, Dercum and Keen, Durante (Case 7), Grisson and Sänger, Jany, Kahler and Pick, Mills and Pfahler, Mills, Keen and Spiller, Mills and Weisenburg (Case 1), Mills and Frazier [133], Monakow ([142] Case 3,

[142] Cases pp. 671 and 687), Oppenheim ([155] Case 2, [156] Case 6, [157] Case 2), Pierret, Roussy, Savill (two cases), Spiller ([192] Case 1), Zeller and Dercum, Stauffenberg (Case 1), Walton and Paul (Case 2), Vetter [224], Westphal. To these thirty cases from the literature I may add three of my own,—viz., the Cases XIX., XX., XXI.

Cases showing very marked and extensive sensory disturbances have, further, been published by Morax and Reymond, but in these cases there seems to have been some additional superposed factor such as hysteria.

Further, numerous cases have been described presenting lesions in the central convolutions only, or in the central and parietal convolutions, in which the lesion has caused definite motor as well as definite sensory disturbances. Here I do not intend to dwell upon these well-known facts.

On the other hand, I wish shortly to touch upon some cases in which sensory symptoms were observed notwithstanding the fact that the lesion at the operation or post-mortem was found in front of the central sulcus—that is, in front of the sensory zone. Cases of this kind have been published by Barker, Beger, Bonhöffer ([14] Case 1, [15] Cases 1, 2 and 3), Dana ([43a] Case 1, [43b] Cases 2 and 3), Grasset, Herbold (Case 1), Hoppe (Cases 5 and 8), Horsley ([90] Case 1), Krause (p. 965), Laycock, Leyden, Mann, Petrina (Cases 1, 3, 4 and 6), Mynter, Nason, Redtenbacher, Starr and McBurney. When Bonhöffer in two cases—([14] Case 1, and [15] Case 2)—and Dana in two cases—([43a] Case 1, and [43b] Case 2)—as well as Horsley, Krause, Leyden and Mann, found sensory disturbances attending lesions of the anterior central convolution, this condition may be easily enough explained by the lesion having been of so great an extent that it might have injured subcortically the tracts of projection of the thalamus passing to the posterior central convolution; with regard to Mann's case, it is even stated that the posterior central gyrus had grown narrower, in spite of the fact that the lesion on the surface did not reach behind to the sulcus centralis. In a majority of cases, however, no account is given as to the size of the lesion.

Still, in the majority of these cases the lesion is of such a nature that it may easily have caused a functional disturbance by means of remote action in the macroscopically or, as in Petrina's cases, even microscopically normal posterior central convolution. In Barker's and in Beger's cases, in Dana's third case ([43b] Case 2), in Hoppe's Case 8, and in Redtenbacher's case we have to deal with a tumour; in

Herbold's case again, in Hoppe's Case 5, and in Nason's case there was an abscess, and in Petrina's Case 6 there was a tuberculous mass. In still another couple of cases, it is true, a vascular lesion was found, but of such a recent date that remote action may be presumed, and the sensory disturbances thus explained. This was also to be suspected in Mynter's and in Petrina's Case 7; moreover, in the former of these cases the hæmorrhage was of traumatic origin. In Starr's and McBurney's case the hæmorrhage had extended into the central sulcus, and direct injury to the posterior central convolution was therefore probable. A similar possibility exists in Bonhöffer's case ([15] Case 1), in which some full veins of the pia mater of the anterior central convolution had been ligated. With regard to Laycock's case and another of Bonhöffer's cases ([15] Case 3), the sources of error, when deciding the topography of the lesion, are too great to allow of any conclusion as to the greater or lesser extent of the lesion posteriorly; and in Redlich's review of Grasset's case no detailed accounts are given as to the extent of the lesion.

Petrina's Cases 3 and 4 are more difficult to explain. Here vascular lesions existed of such an old date that no initial diaschisis can be assumed as the cause. But as in several cases showing similar lesions of the parietal region motor disturbances were found in spite of this function being localized to the anterior central convolution, it seems also justifiable in Petrina's cases to refer the sensory disturbance to a remote action.

On the other hand, Nothnagel and Mills have found that lesions localized to the anterior central convolution may run their course without giving rise to sensory disturbances. This is also Monakow's and Oppenheim's experience, and cases pointing in the same direction have also been related by Bergmann (*cit.* Vetter), Redlich [169], Long [119], and Förster [68]. I myself have had the opportunity of observing such a case, in which the diagnosis was proved at the autopsy by means of histological examination. In two cases—Dana ([43b] Case 3) and Hoppe (Case 6)—of tumour in the gyrus centralis anterior there was at first a pure motor paresis, and the sensory impairment (which in Dana's cases was only transient) appeared after trephining.

From this it is evident that lesions of the centro-parietal region often give local sensori-motor symptoms, but at the same time where the lesion was situated behind the central sulcus, the sensory symptoms may predominate. Study of the literature shows that this is the case only in lesions localized in this position. Further, it is evident that in connexion with lesions of the anterior central convolution sensory disturbances may be absent, and finally that those cases showing sensory disturbances in connexion with lesions affecting the anterior convolution, or parts situated still further forward, do not entitle us to presume a sensory function in these parts; for the sensory disturbances in all these cases, with the exception of two, may be explained by the size of the lesion or through remote action. The clinical and anatomical facts thus agree with the results of experimental and histological investigations of recent years, that a common sensorimotor area does not exist in man.

(b) *The Extent of the Sensory Projection-Area.*

Clinical experience thus shows that motion and sensation have different localization. It also shows that sensation is localized behind motility, but when it is a question of drawing the boundaries of the sensory projection our knowledge is less certain. We are, it is true, able to draw with great accuracy the anterior boundary for the sensory projection somewhere near the central sulcus, but not without a certain reservation for individual variations on account of Sherrington's discovery with regard to the boundaries of the motor area. That the border of the sensory projection cannot lie behind the posterior central convolution, and that this, consequently, is a part of the sensory projection, is proved by numerous cases that have been investigated in detail with regard to sensation, where a lesion has been situated either to the central convolution alone, as in the cases of Krönlein, Knapp [102], Gray, Grissom and Sänger, Monakow ([141] p. 671), and Horsley and Russel (Case 3), or to both the central convolutions without having gone beyond them posteriorly. Cases of this kind have been published in great numbers, and I will here draw attention to the cases by Dana, Edinger, Friedländer and Schlesinger, Gros, Bramwell, Horsley and Russel, Wernicke, Wilson, Verger, Verger and de Boucaud, Petrina, Levi. In these cases obvious sensory disturbances existed, and the anterior central convolution being motor, not sensory, it is evident that the lesion occupying the posterior central convolution in these cases was the cause of the sensory disturbance.

On the other hand, the posterior boundary of the cortical projection can be defined with less exactness. It may, however, be said to extend posteriorly beyond the posterior central convolution, and the opinion expressed by Long and Henschen, to the effect that the sensory projection posteriorly does not extend beyond the posterior central gyrus, is not sufficiently supported by their own cases of cortical lesion, all of which presented foci involving in addition the parietal lobe. Further, several cases have been published of lesions occupying the parietal lobe attended by sensory disturbances. Thus in the majority of cases previously quoted, where the sensory disturbances appeared earlier or even predominated in comparison with the motor disturbances, the lesion occupied the parietal lobe exactly. Monakow's case ([142] Case 3) showed marked disturbances of the muscular sense from a lesion of the medulla of the angular and supramarginal gyri, although both central convolutions, together with their tracts of projection, were microscopically normal. This, together with Stauffenberg's recently published case, proves that the parietal lobe possesses sensory function. And in a great number of cases which, during an early stage of the disease, showed grave motor disturbances, in addition to the sensory disturbances, the lesion occupied the parietal lobe. Cases of this kind have been published by Henschen ([85] vol. i, Case 18; vol. iii, Cases 6 and 11; vol. iv, Cases 2 and 12), Long ([118] Cases 3 and 5), Madden, Souques, Wernicke and Hahn, Stewart and Vetter. These cases are, however, less convincing than the former ones, as the presence of motor disturbances clearly points to an obvious remote effect on the part of the lesion, which naturally must have influenced not only the anterior but also the posterior central convolution and the respective tracts of projection.

Definite support for the sensory function of the parietal lobe is furnished by Monakow [141], who found degeneration of the lemniscus in connexion with purely parietal lesions. It would thus seem to be obvious that at least certain parts of the parietal lobe are provided with sensory function.

But it seems doubtful whether the whole of the parietal lobe has sensory function. Gordon [73], for instance, found in a case of gunshot injury, followed by considerable destruction of the parietal lobe, that sensation was fully intact, but, unfortunately, no further information is given with regard to the extent of the lesion in this case. In the previously quoted case by Mills and Frazier [133], where a benign cyst occupied the angular gyrus, the sensory symptoms had such a distribution and were so slight that one is entitled to presume they were due to remote action and not to the lesion itself, still more so as they completely disappeared after the operation. Not even disturbances of the muscular sense always appear in connexion with small lesions of the supramarginal and angular gyri, according to Cornil, Jastrowitz, and Monakow (cit. Monakow, [141]).

On this account it is probable, as shown by Mills on his diagram ([119] p. 333), that the whole parietal lobe is not provided with sensory function, or at any rate that the sensory function is not equally marked in the whole lobe; and even if at present it is impossible to define the posterior boundary of the sensory projection-area in man, still, on the other hand, it is certain that the anterior part of the parietal lobe has sensory function.

(c) *Subdivision of the Sensory Projection.*

Just as the motor projection is divided into subcentra for the movements of different parts of the body, so the sensory projection, as has been pointed out by Mills and Weisenburg, is divided into subcentra for the sensation of different parts of the body. From cases from the literature and from those under their own observation these authors have proved the possibility of a partial sensory disturbance following a cortical lesion affecting, for instance, only one or a couple of fingers. When giving an account of the distribution of the cortical sensory disturbances I was able to add a number of cases from the literature, together with a few of my own, to those related by Mills and Weisenburg, which demonstrated a partial distal sensory disturbance. But besides this partial distal type, a few cases are also met with showing disorders of sensation more marked proximally. Here a similarity is found between the distribution of the motor and sensory disturbances of motility, often of different intensity in different parts of the hand, and cases also exist, though less commonly, in which the paresis is less marked distally than proximally.

Mills and Weisenburg point out that the paralysed extremity also presents the greater sensory disturbance, an observation of the same kind as the one advanced twenty years ago by Henschen against the doctrine of "Carrefour sensitif." But, whereas Henschen and others drew the conclusion that motility and sensation had the same projection, Mills and Weisenburg drew the conclusion that motion and sensation of one and the same part of the body stand in a close topographical relation to each other, are situated "at the same horizontal level," but not that they coincide with one another (a doctrine against which Mills has long argued). The sensory functions which Mills and Weisenburg found to be affected had reference both to the cutaneous and muscular senses. But the disturbance of cutaneous sensation is not constant in these cases of partial distal sensory disorder, as is obvious from cases selected from the literature and from my own observations narrated above. Amongst these there were several showing an affection of the muscular sense only. The fact that Mills and Weisenburg found stereognostic perception also affected to the same extent as the cutaneous and muscular senses is of less interest with regard to the division of sensation into subcentra; for, as pointed out by Dejerine [51], a disturbance of the more simple function is followed by a disturbance of the complicated function into which the former enters as a component.

(d) *The Cortical Localization of the different Sensory Functions (the Cutaneous and Muscular Senses).*

(a) *Simple and Associated Functions.*

If, then, sensation has a widely distributed area of projection, and since it is possible to distinguish physiologically different functions of sensation, we may presume that these different functions of sensation have different areas of projection on the cortex.

As, further, Campbell has proved that undoubted histological differences between different parts of the large sensory area of projection certainly exist, this presumption gains in strength. But even though we cannot draw any direct conclusions with regard to function from these structural differences it is justifiable to suppose that difference in structure is associated with functional differences. And when Campbell found that the anterior part of gyrus centralis posterior, but no part of the sensory area behind it, showed *réaction à distance*, after capsular hemiplegia and amputations, this indicates, as he points out, that the anterior part of gyrus centralis posterior stands in closer relation to the periphery than do parts situated more posteriorly. Roussy's discovery that the thalamic fibres ran specially to the posterior central convolution also points to a functional difference compared with the parietal lobe. Campbell is of the opinion that it is "common sensations" that are localized to the post-central area proper, whereas it seems as if he were inclined to locate the muscular sense to the intermediate post-central area ([35] p. 306).

It might, however, be objected that, even if it is possible to differentiate between different centres with regard to their closer or more remote relation to the periphery on the one hand, and on the other to make a division of the functions into simpler and more complicated ones, we are not entitled to place on the same level a definite centre and a definite function.

Sensory functions may be simple or complex; for instance, the perception of a touch pure and simple is a less complicated function than perception of touch and its localization. The latter is an associated function which is most likely composed of more components than the single sense of touch only, judging from the fact that disturbances of localization have been discovered in association with disturbances of now one and now another function. Disturbance of any component of a complicated function ought to lead to a disturbance of the function itself (Dejerine [48, 51], Morton-Prince). It might be possible that a disturbance of one component could be compensated to a greater or lesser degree, but in order to prove with certainty the existence of a limited local centre for a complicated function, disturbance of its components must be excluded, obviously in many cases a difficult task.

But, by examining the tactile sense and the power of localization, it is possible to compare the condition of a complicated function and one of its most important components.

With the perception of passive movements this is not possible. It is possible to compare the perception of movement as such with the perception of the direction of the movement; but, as Nagel points out, even the former function is to be considered as a complicated one, composed of sensations from different organs. We do not know with which organs these sensations are connected, and the components which enter into the perception of passive movement are at the present inaccessible for clinical investigation; but disturbances in these components ought none the less to cause a disturbance in the perception of passive movement and position.

In its turn this function, the perception of movement, no doubt plays the part of a component, and is of the greatest importance in the case of the highly developed functions co-ordination and stereognosis; the relation between the muscular sense and stereognosis thus becomes analogous to the relation between tactile sensation and power of localization. But many other functions are included as components, among others possibly the function of motility. For this reason we should expect that the more complicated functions would show disturbances more often than the simpler ones as the result of cortical lesions, and this is fully confirmed by clinical experience.

The functions on which I have concentrated my attention are the cutaneous senses and one function of the muscular sense—viz., the perception of passive movement—and I have tried to obtain a conception of their localization within the sensory zone on the basis of cases from the literature and from my own experience.

(β) *The Localization of Cutaneous Sensibility.*

In the case of lesions within the whole of the centro-parietal region the simple cutaneous senses have been reported to have been disturbed, especially in recent investigations. This is so obvious that I shall only refer to Dana's [43] and Long's [118] investigations with regard to this point.

In connexion with lesions within the central convolutions alone, one finds statements as to disturbances of the cutaneous senses. Unfortunately, a microscopical examination has not been made, and the boundaries of the lesion cannot be said to have been established with certainty. Such is the case in the cases by Albertoni and Brigatti, Dana ([43a] Case 4, ([43b] Case 1), Darkschewitsch, Friedländer and Schlesinger, Horsley ([89] Case 1, ([90] Case 2), Horsley and Russel (Case 2), Verger and de Boucaud, Wernicke (Cases 1 and 2), Walton and Paul ([217] Case 2), and Grisson and Sänger. And of special interest in this connexion are the cases of small lesions which have been published by Gros, Levi, Gray, C. Knapp [102], Krönlein, Monakow ([141] p. 671), and Thomas.

Disturbances in the simple cutaneous senses have also been observed in connexion with lesions in the gyrus centralis anterior, which have not directly attacked the sensory zone, at least macroscopically. But even in cases where the lesion has been situated in the parietal lobe without affecting the central convolutions macroscopically, or at the autopsy *in vivo*, one often finds definite statements that disturbances, though slight only, were present in cutaneous senses. (Bruns (Case 2), Cornil, Durante (Case 8), Henschen ([85] vol. III, Cases 6 and 11; vol. IV, Case 1), Mills, Keen and Spiller, Mills and Weisenburg (Case 1), Mills and Frazier [133], Mills and Pfahler, Oppenheim ([154], [156] Case 6, and [157] Case 4), Petrina (Case 5), Pierret, Roussy, Souques, Stauffenberg (Case 1), Teller and Dercum, Westphal, and Vetter.)

In a great majority of these cases no microscopical examination was made, and in many of them a remote lesion may be suspected; but still we may with certainty presume that lesions situated either in the central or parietal convolutions may cause disturbance in the simple cutaneous senses.

But the disturbances of the simple cutaneous senses are not constant in connexion with these lesions. The absence of disturbances of simple cutaneous sensations in connexion with local lesions of the central convolutions is mentioned in a case by Diller (Case 1, in which tabes was also present), further by Hoppe (Case 4), Horsley ([90] Case 2). The same applies to a lesion within the parietal lobe, where the simple cutaneous senses are stated to have been normal in cases related by Gordon [73], Oppenheim ([155] Case 2, [156] Case 5), Starr and McCosh, Stewart (where disturbance of simple cutaneous senses was found only in connexion with an operation), as well as Monakow ([142] Case 3), where the posterior central convolution at a microscopical examination did not show any exchanges. Even if the lesion occupies almost the whole centro-parietal region, as in a case of Hoppe's, it may happen that the cutaneous senses are unaffected. This, however, would seem to be an exception.

With regard to my own observations, Cases XIX. and XX. are of interest in connexion with the question of the localization of cutaneous sensation. The extent of the lesion was in both cases very much alike. It occupied the greater part of the parietal lobe, as well as the posterior part of the gyrus centralis posterior (microscopical examination on serial sections); but in the former case the lesion was 1¼ year old, in the latter case it was of a recent date. In the former case the simple cutaneous sensations were only slightly, in the latter greatly, affected. The reason why two cases with lesions so similar from an anatomical point of view have presented such different symptoms can with certainty be looked for in the presence of an initial diaschisis in the latter case, and to a secondary restitution in the former case; the cases thus do not contradict the presumption that the simple cutaneous senses are localized to the anterior half of the gyrus centralis posterior, but they do not determine whether this is the only localization. With regard to the localization of simple cutaneous sensations, no knowledge can be obtained from the cases quoted above from the literature except that the cutaneous senses have certainly some representation in the posterior central convolution, and that they are also most likely represented in the parietal lobe. When it is a question of the nearer details of this localization, the accounts to be obtained from the literature are contradictory, and it is difficult to imagine a sharply defined focal projection for the simple cutaneous senses of the same kind as exists in case of motility, more especially when we consider how completely disturbances in the simple cutaneous senses may ultimately pass away.

This power of repair of the sensory functions is at the present a complete puzzle, showing as it does an apparent capriciousness, and all explanations up to the present time are mere hypotheses. Henschen and Müller ascribe this power of reparability to the healthy hemisphere;

Monakow [141] is of opinion that the simple cutaneous senses of each part of the body are represented in a number of gyri; Cajal [34] considers that the repair takes place through a real regeneration of destroyed elements, without mentioning any investigation in support of this theory.

The power of localization, being an associated function, possesses the power of restitution to a much less degree, and may also present disturbances at an early stage of focal lesion, without any disturbance of tactile sensation. This may occur whether the lesion occupies the central or the parietal convolutions. (Bonhöffer [15] Case 1), Hoppe (Case 6), Starr and McBurney, the lesion in these cases occupying the central convolutions; as well as Stewart, Walton and Paul ([217] Case 4), where the lesion also affected the parietal lobe.)

Disturbances of the power of localization were also found in those cases where tactile sensation was reduced.

Considering the connexion existing between disturbances of a simpler and a more complicated function, it is evident that such cases do not support a focal projection of the power of localization on the cortex.

Still, the power of localization being an associated function, disturbances of it should be compared with disturbances of some other associated function, and not only with those of the "simple" cutaneous senses.

(γ) *The Cortical Localization of the Muscular Sense (the Perception of Passive Movement).*

The functions associated with the muscular sense are, as a rule, those that present the greatest disturbances with cortical as well as capsular lesions, and may even be the only ones affected. That this is the case with regard to the stereognostic power and symbolic recognition by touch is generally known.

I do not consider myself entitled to discuss the disturbances of this function in detail, as they lie beyond the limit of my investigation. The disturbances of muscular sense, which I have made a subject of closer investigation, are those which we generally have in view when speaking of the muscular sense—viz., disturbances that have reference to movement and position. These functions are, as a rule, affected in the case of lesions of the sensory zone. They may be affected without the simple cutaneous senses showing any disturbance. In the case just mentioned, of simultaneous disturbance of the function of the muscular sense and of the simple cutaneous senses, the disturbances of the former function were much more extensive than disturbances of the latter, and in the majority of cases this occurs whether the lesion is situated in the anterior part of the sensory zone or in its posterior part.

A dissociated sensory disturbance is recorded in many cases where the lesion occupied the central convolutions, or the anterior central convolution—i.e., in cases where the lesion was situated either in the anterior part of the sensory zone, or affected it from in front. In two of these cases only—viz., Grisson and Sänger's, and Starr and McBurney's—is it mentioned that the muscular sense was not affected, but that the cutaneous senses, on the other hand, were disturbed (in Starr's and McBurney's case, however, only the power of localization). These cases would thus indicate that the muscular sense may be unaffected in spite of a disturbance of the cutaneous senses when the lesion from in front has affected the sensory zone.

This is, however, not the rule; in several of the cases where the lesion occupied the anterior convolution, and where definite statements with regard to this point are given, the muscular sense proved to be more affected than the cutaneous senses, Bonhöffer's cases ([14], [15] Cases 1, 2 and 3), Hoppe's (Case 6), Laycock's, and, lastly, Krause's case with ataxia, where the lesion was also situated in the anterior convolution, and yet the disturbance of the muscular sense was the most marked feature. Of special interest is Mann's case, where, in connexion with Jacksonian epilepsy and especially after the fits, the greatest disturbances of the muscular sense were present, but during the residuary stage, affection of the muscular sense was less conspicuous than other sensory disturbances.

Thus in several cases where the lesion from in front affected the sensory zone the disturbances of the muscular sense were those most prominent. The same applies to several cases where the lesion occupied the anterior part of the sensory zone: Bonhöffer ([15] Case 4), Edinger [59]. Horsley ([90] Case 2), Kronlein, Levi, Monakow ([141] p. 671), Murarow, Oppenheim ([156] Case 5, [157] Case 2), Gray, Knapp [102]. The same thing prevailed in my own Case XVII., in which the paralysis by degrees spread to the sensory region.

From a study of the literature I have, however, obtained the impression that it is especially in connexion with lesions occupying the whole of the centro-parietal region, or the posterior part of it, that disturbances of the muscular sense predominate. Cases where the lesion has occupied the whole of the centro-parietal region are, however, naturally less suitable for the study of localization of the muscular sense. On the other hand, those cases are of value which show a lesion in the posterior part of the sensory zone with predominant disturbances of the muscular sense; such cases have been described by Binswanger (Case 3), Bruns (Case 2), Cadwalader, Dercum and Keen, Herbold (Case 2), Hudson, Mills and Weisenberg, Mills and Frazier [133], Mills, Keen and Spiller, Monakow ([141] p. 687, [142] Case 3), Oppenheim ([154], [155] Case 2), Roussy, Souques, Spiller, Starr and McCosh, Stewart, Walton and Paul ([217] Cases 2, 3 and 4), Westphal, Wernicke and Hahn, Vetter, as well as my Cases XIX., XX., and XXI. In all, the disturbances of the muscular sense were the most prominent sensory affection; and in several of the cases disturbances of the cutaneous senses were entirely absent (*vide* p. 451). In this respect Henschen's cases are especially contradictory, but, as the method employed by him is not sufficiently definite and as his investigations have not been directed especially to this point, I do not consider that in this respect any decisive importance can be attached to them.

This has been taken to mean that the centre for muscular sense is situated in close relation to that for motility (Wernicke, Bonhöffer and others), that muscular sense is situated in the parietal lobe (Mills and others), or that muscular sense is situated in both these places (Monakow).

When we have to deal with that function of the muscular sense that is called stereognosis (symbolism), it is easy to explain why it is so often affected as to be (Monakow) a remarkably fine reagent for cortical lesions. This function is of a very complicated nature; into it enter not only cutaneous senses and the simpler functions of the muscular sense, such as perception of movements and position, but also motion. For, as Markowa points out, a disturbance of stereognosis may result with disturbances of motility.

The same applies to co-ordination, and with regard to the orientation in space I have tried to show this in the preceding pages. This function was affected partly in connexion with disturbances of the muscular sense and partly in one case in connexion with disturbances of motility. With regard to the power of localization and the "sense of place," these functions may be affected in connexion with disturbances of one of their component functions, as, for instance, tactile sensation, but may, on the other hand, be disturbed without any signs of affection of tactile sensation. In the majority of cases the sense of position and movement and the power of localization are simultaneously affected. Thus the complicated functions, unlike the simple cutaneous senses, are affected alone or to a preponderating extent whether the lesion is situated in the anterior or posterior part of the sensory zone. But in cases where the lesion does not directly attack the sensory zone the same condition may occur through remote action, as is evident from my Cases XVII. and XXI., as well as from Mann's and Binswanger's experience.

In my Case XVII. we have to deal with a lesion (an abscess) which at first only gave motor symptoms of irritation and lost function. At this time the lesion was limited to the motor zone; later, after convulsive attacks, sensory disturbances appeared, which to begin with were of a transient post-paroxysmal nature. These transitory sensory disturbances consisted of intense disturbance of the perception of movement and position with minimal disturbances only of the cutaneous senses; these transitory symptoms receded so quickly that at this time they cannot possibly have been due to organic destruction. During this recession the perception of movement and position was affected for a longer time than the cutaneous senses. When, later on, as the lesion progressed, lasting sensory disturbances appeared, those of the muscular sense were again most definite. In Case XXI., on the other hand, due to a lesion in the posterior part of the temporal lobe, the sensory disturbances developed at a late period of the disease, and the perception of movement was gravely affected, although there was a suggestion only of loss of tactile sensation on the other hand. In this case transient sensory disturbances, particularly of the muscular sense, appeared in connexion with the attacks.

We thus have two cases in which the sensory disturbances developed in connexion with processes which are generally followed by remote action; they appeared in connexion with conditions which generally increase remote action, conditions which, after the fit has passed off, quickly diminish, thus showing themselves to be of functional, not of organic, nature. In the former case the remote action extended from in front, in the latter from behind and below, on to the sensory zone; in both cases the disturbance of the muscular sense was well marked, whereas in one case only the tactile sense showed any disturbance in the hand.

Now, however, the question presents itself whether the complicated nature of the muscular sense does not cause it to be affected to a higher degree than the cutaneous senses through a remote effect (diaschisis) in connexion with cerebral processes of any kind. By diaschisis Monakow [141] means a kind of paralysis due to a loosening of local contact, dissolution especially at the transition of one neuron chain to another (eine "Lähmungs erscheinung hervorgebracht durch eine lokale Kontacktlockerung oder dgl. und zwar an der Uebergangsstellen einer Neuronenordnung in die nächstfolgende"). According to Monakow, a return of the function should take place through "Neugliederung" and "Neueinübung."

If one applies this hypothesis of diaschisis to disturbances of the muscular and cutaneous senses following cortical lesions, it seems not unlikely that the more complicated function in these cases, the perception of movement, which may be presumed to be composed of several neuron-chains, should suffer more than the cutaneous senses, simpler functions which may be presumed to consist of less numerous neuron-chains. This view might possibly explain why the muscular sense, as a rule, is found to be more affected than the cutaneous senses in connexion with cortical lesions, whether they attack the sensory zone from in front or behind. But, on the other hand, this view considerably reduces the value of those cases which at present serve to support a limited localization of the muscular sense.

The material from which the cases I have quoted are taken is naturally very heterogeneous. Thus it is clear that from the cases hitherto published it is only possible to draw conclusions with regard to the localization of the muscular sense within the sensory area with a certain degree of reservation. This reservation becomes so much more justifiable if, as my Cases XVII. and XXI. seem to indicate, the simpler and the more complicated functions were to show a reciprocal difference with regard to diaschisis. Still, it is possible that the two cases of Grisson and Sänger and of Starr and McBurney, where the muscular sense was unaffected, in spite of disturbances in the cutaneous senses, may indicate that the muscular sense is situated behind the cutaneous senses. There is no support for a more detailed localization or circumscribed focal projection of these functions. Nor can we find any definite support for a circumscribed focal projection of the cutaneous senses, but there are strong reasons for believing them to be at least partly represented in the posterior central convolution.

It may, on the other hand, be regarded as proved that the projection on the cortex of the motility and sensibility are separated; that the latter is situated behind the former; and that it occupies the posterior central convolution and at least the anterior part of the parietal lobe; lastly, that within this sensory zone a subdivision exists for different parts of the body, as is the case within the motor zone.

In brain-physiology the connexion between sensory function (especially that of the muscular sense) and motor function has been the subject of numerous speculations and experimental investigations. Several physiologists are of the opinion that the motor symptoms of lost function are a consequence of the sensory disturbances. In order distinctly to emphasize the importance of sensibility, Munk has changed the name "motor zone" for "Fühlsphere," and in the paralysis appearing after extirpation of the Rolandic region he sees a psychical disturbance, consisting in the loss of motor perceptions. An analogous standpoint is held by Bastian.

The facts here brought forward seem to speak decidedly against Munk's doctrine. From them it is evident, firstly, that motility may be affected in spite of integrity of such highly associated sensory functions as the muscular sense, stereognosis, and symbolism. Besides, I have shown that orientation in space can be unaffected in spite of evident paresis.

Munk's view also stands in opposition to those cases where sensation, and more especially the various functions of the muscular sense, have been affected without the presence of any defect of motility.

Consequently, since disturbances of the one or the other functions can appear alone, Munk's view cannot be applicable to man. The problem as to the significance of the motor conceptions for motility seems to me to lie beyond the scope of this essay; with regard to this question I shall only call attention to a case recently published by Förster [68] which seems to indicate that it is not movements only that are represented in the cortex, but that different muscles also have their projection area on the cortex, a view entirely at variance with Munk's opinion.

CHAPTER VI.—THE SPASTIC PHENOMENA IN CORTICAL LESIONS.

§ 1.—*Babinski's Sign.*

When my cases are examined with regard to the occurrence of spastic phenomena, hypertonus, contracture and reflex hyperexcitability, it is striking how little marked they were in the cortical as compared with the capsular cases. The character of the spastic phenomena in cases of cortical and capsular lesions does not seem to have been the subject of any thorough investigation in the recent literature, except with regard to the occurrence of Babinski's sign.

This reflex consists, as is well known, in a dorsal flexion of the great toe, on stroking the sole, instead of plantar flexion. It occurs normally, while awake, during the first three years of life in rickety children, and during sleep still longer (Pfaundler); but beyond that age it points to a lesion in the central motor tract. This is, as a rule, the earliest sign of a beginning spasm, and often shows itself when in all other respects the limb is completely hypotonic. Undoubtedly, however, there are cases in which Babinski's reflex is not met with, in spite of the existence of a cerebral paralysis (Ganault, Græffner, Prohazka, Goldflam). This not only occurs in lesions affecting the basal ganglia or the brachia (Oppenheim [158]), but also, according to Bychowski, in connexion with lesions situated in or close to the cortex; in Spilmeyer's singular case of intracortical hemiplegia this sign was lacking, in spite of an increase of the tendon-reflexes and the presence of contractures.

In my cases Babinski's sign did not appear in 7 out of 18 cases of cortical disease—viz. in VI., VII., VIII., XI., XII., XV., XVIII. As regards Case XXIII., on the other hand, which will be related later on, the sign in question was at first present, then vanished during the contracture period, but afterwards reappeared again. In four cases Babinski's sign was present—viz. in IX., X., XIII. and XIV., whereas in the remaining five cases it was doubtful. Thus in a considerable percentage of this small collection of cortical lesions the Babinski reflex was lacking.

The practical significance of the fact that Babinski's sign is often absent in cases of cortical lesions is obvious, considering that the absence of this sign in a paralysis of the cerebral type has been considered to denote that it is of functional origin.

§ 2.—*The Tendon Reflexes.*

As is well known, an increase of the tendon reflexes and a condition of hypertonus of the muscles generally occur in most cases of supranuclear lesions. In Monakow's account of this question in his "Gehirnpathologie" (p. 537) exaggeration of the reflexes is put on a par with a hypertonic condition of the muscles as being a lower degree of the same process. Hence the conditions which hold good with regard to the appearance of the higher degree, hypertonus, also ought to be valid with regard to the occurrence of the lower degree, excess of the reflexes. On the other hand, it is evident that excessive reflexes may occur without hypertonus being present, or before it has had time to develop. The time at which the reflex hyperexcitability develops in a hemiplegia varies considerably, but according to Monakow and Oppenheim it comes on as soon as the patient wakes up from the comatose state. Contractures seldom show themselves before the end of the second week, usually between the sixth and twelfth week (Dejerine), and are always accompanied by an increase of the tendon reflexes. In all my twenty-five cases of typical capsular hemiplegia the spastic symptoms were definite, and reflex hyperexcitability was present as well as hypertonus.

During the initial limp paralysis, which is considered by Monakow to be due to diaschisis, an increase of the reflexes does not occur as long as the diaschisis period lasts, as a rule merely a few days. In tabes, Oppenheim [158] has shown that an exaggeration of the reflexes may be totally lacking. To this complication Oppenheim attaches great importance, and, according to his experience, such a case of hemiplegia, without excessive reflexes, frequently turns out to be a tabes, even though a tumour has been the cause of the paralysis. Graffner has, furthermore, arrived at the conclusion that in diabetes and in marasmic conditions, due, for instance, to pernicious anæmia or pulmonary tuberculosis, the reflexes may be totally absent, or, at any rate, no marked increase of the reflexes may occur after a hemiplegia. The very brief form of his statement makes it, however, impossible to decide whether in those few cases the result has not been influenced by some other circumstance. In cases of tumours and abscesses it frequently happens, as Sternberg has pointed out, that no increase of the reflexes takes place, and the very large number of reports of cases of this kind that have since appeared has fully borne out his statement, both with regard to capsular and cortical tumours. In two cases Reh was able to show that the reflexes disappeared during periods of increased intracranial pressure. But in four other cases amongst those collected by Reh, where the reflexes were permanently lacking, changes in the posterior columns were found. According to Collier and Batten, degeneration of the posterior columns occurs in most cases of cerebral tumours, but its intensity and extent is so insignificant (Petrén) that it can scarcely be thought of as an explanation, the more so as such degeneration has been also found in connexion with vascular lesions attended by an increase of the reflexes (Guillain). Further, it sometimes happens that in infantile hemiplegia the reflexes are lacking (Long and Noica). According to Mills [129], some spinal complications should always be suspected in such a case. With regard to one case, Long [119] is of opinion that the absence of increased reflexes may be put down to a lesion that had originated so early (intra-uterine?) that the pyramidal tract was not yet developed.

The locality of the lesion may also be of importance independently of its character. Thus, it is well known that in lesions of the cerebellum, &c., the knee-jerks may be wanting. But this is a question which does not directly concern us here. According to Williamson, this holds good also with regard to affections of the frontal lobes; but, as the cases upon which he bases this statement consist of tumours and abscesses, it coincides with what Sternberg has found.

By some such complication it is possible to explain why the reflexes are not exaggerated in some particular case of supranuclear lesion. But I have found vague allusions only to the fact that the cortical seat of the lesion may in itself be sufficient cause for this absence of exaggeration of the reflexes. Oppenheim, for instance, in the last edition of his text-book, mentions that in monoplegia hypotonous occurs exceptionally, but he does not say whether he refers to tumours or vascular lesions.

Monakow [141] is the only author, as far as I have seen, who gives a somewhat more detailed account of the varying extent to which hypertonus is present in different cerebral affections; and as he considers that reflex hyperexcitability is a lower degree of the same process, I may be justified in recording his views in this connexion. According to Monakow, the hypertonic state becomes more marked the nearer to the spinal cord the lesion lies within the motor path, and the more completely the motor tracts have been destroyed; further, cortical or subcortical ataxy may be attended by a hypotonic condition, whereas more extensive lesions within the motor zone are followed by contractures. It should, however, be observed that the hypertonus becomes less marked if disorders of sensation exist at the same time.

Dejerine [48] and Strümpell [205], on the other hand, are of opinion that cortical and capsular lesions behave in an exactly similar manner as far as the hypertonic state is concerned, and Mills [129] believes that, if the reflexes are not increased in a case of hemiplegia, an extra-cerebral affection should be suspected.

Passing on to my own cases, it will be found that all those with capsular lesions (except Case V., of which more below) presented marked hemispastic symptoms. But amongst my cortical cases an increase of the reflexes were present in ten instances—viz., in Cases VI., IX., X., XII., XIII., XIV., XVI., XIX., XXII., and in Case XXIII. (described further on in this chapter), but only in Cases IX. and XVI. was the increase comparable with that usually met with in a typical hemiplegia. The increase was, however, strange to say, greater in that extremity which was not paralysed. In six cases—viz., VII., XI., XV., XVII., XVIII., and XX.—the reflexes were equal on both sides. In two other cases the reflexes were even weaker on the paretic side—viz., in Cases VIII. and XXI.

In Case V., which belonged to a transitional type between a cortical and a capsular lesion, the reflexes of the legs were excessive, those of the arm diminished; even in Cases XIV., XVI., XXI. and XXIII. such a difference existed, though less strongly. In these five cases the arm alone was paretic or more so than the leg; in Case IX., again, where the leg presented the highest degree of paresis, there was present an exaggeration of the reflexes of the arm, but not of the leg.

The contrast between the cortical and the capsular cases is striking. Can this contrast be explained by the existence in such cases of some circumstance alleged in the literature to be the cause of the non-occurrence of the typical increase of reflexes? This could have been the case, however, with regard to a small minority only. I need not dwell upon the fact that several of these cases, cortical as well as capsular, when they first came under observation, did not present any exaggeration of the reflexes, but showed an initial limp paralysis, which afterwards disappeared. Further, in Cases VI., XVII., and XX. the paralysis was due to an inflammatory lesion, and in Case XXI. to a tumour—i.e. to processes which often occur without any increase of the reflexes at all.

A complicating process might perhaps have been of some influence in Cases VIII. and XIV. In the former, metasyphilis was certainly present. In the latter there was also syphilis in the patient's previous history, and he had a right-sided hemiplegia and a left-sided hemiatrophy of the tongue. True, this latter symptom may have been due to some other lesion than that which had caused the hemiplegia, but, on the other hand, it also occurs in some rare cases of tabes. No other reason to assume tabes existed in this case, and, moreover, the reflexes were increased in the lower extremity. Still, on account of Oppenheim's experience, these cases must be looked upon as less convincing. In Case XV., on the other hand, we have to deal with considerable alcoholic excess; but, considering the fact that in the common cases of capsular hemiplegia in alcoholic persons the normal exaggeration of the reflexes appears as usual, we are justified in neglecting this complication. That in Case XII. there is reason to suppose that the lesion had attacked the frontal lobe is of no importance, as the lesion was evidently of a vascular nature. Moreover, as distinct motor paralysis existed, it must have attacked the motor zone also; hence there is no reason to discard this case.

If I eliminate Cases VI., XVII., XVIII., XX., and XXI. on account of the nature of the process, and Cases VIII. and XIV. on account of the presence of a possible complication, nine cases of cortical lesion still remain where the explanations previously recorded in the literature are not sufficient to account for the non-appearance of the usual reflex hyperexcitability. In three of these cases—VII., XI., and XV.—the reflexes were equal on both sides; in six—X., XII., XIII., XIX., XXII. and XXIII.—they were slightly increased; in two Cases only—IX. and XVI.—was the exaggeration of the reflexes comparable with that which usually occurs in ordinary hemiplegia, but it was most marked in the extremity which was less paralysed. Of these Cases XIX. and XXII. showed mainly sensory signs.

Case V. is of interest in this connexion, for the anatomic alexamination shows that cortico-vascular lesions need not give rise to hyperexcitability of the reflexes. The case presented a limp paralysis of the arm with loss of reflexes; the leg was not paralysed, but highly paretic and the seat of hypertonus with exaggerated reflexes. The onset dated eight months back, and the paralysis was residuary. Post-mortem there was a vascular lesion, which had destroyed the centre for the arm and its paths of projection cortically and subcortically down to the basal ganglia. The cortical centre of the leg was, on the other hand, uninjured, but its paths of projection had been affected just above the basal ganglia. This case seems to me to suggest, with great probability, that, with regard to the behaviour of the reflexes, it is of importance whether the centre in the cortex is affected or not, or, in other words, that lesions which destroy the cortex may give rise to a paralysis without spastic symptoms.

Monakow has referred to a certain connexion between the degree of paralysis and the degree of reflex hyperexcitability. As far as a comparison between different cases is concerned, my cases give no support to such a supposition: in Case XII., exaggeration of the reflexes appeared with slight paresis; in XV., normal reflexes existed, with considerable paresis; in XI., the reflexes were equal on both sides, but somewhat brisk, and the paresis was slight; in X. and XIII., the reflexes were slightly increased, with complete paralysis; in XIX., the reflexes were slightly increased, but there was no paresis.

But even if we compare the degree of paresis and the degree of reflex augmentation in different extremities of the same patient, no parallelism can be discovered in my cases; on the contrary, the four cases where any difference was evident presented the opposite condition—that is to say, the limb which was not paretic gave a livelier reflex (Cases IX., XII., XVI. and XXIII.). This circumstance, which seems to me remarkable, might perhaps be explained in the manner indicated by the anatomical examination of Case V.—viz., a cortical lesion in the centre for the extremity which did not show excessive reflexes, a subcortical lesion for that limb in which the reflexes were increased.

In this connexion it must first be pointed out that the lesion must have been situated in the focus for the most affected extremity, whilst the other extremity was only affected to a smaller degree. Further, a slight lesion of the motor tract may manifest itself, not by means of a spastic paresis, but through a spasm pure and simple, as may also be the case after a slight hemiplegia (Mann [123]). This fact is well known from the spastic spinal paralysis, where the spasm may long precede the paresis. In the case of a vascular lesion occupying the centre for the arm, this difference between a paralysis of the arm and a spasm of the leg is easily accounted for, as the same artery supplies not only the cortical centre for the arm, but also the internal capsule and thus the tracts of projection of the leg, but not the cortical centre for the leg (Beevor). It is, therefore, unnecessary to have recourse to remote influence in order to explain the fact that the paralysis gravely affected the arm, the cortical centre of which was greatly damaged, whilst the leg, the tracts of which have only been affected subcortically, showed increase of the reflexes only. This circumstance is accounted for by the fact that the leg, in contradistinction to the arm, has different arteries for its cortical centre and its paths of projection.

Where, on the other hand, a vascular lesion affects the centre for the leg, as in Case IX., the matter is somewhat different. Any remote influence ought to assert itself along the border of the lesion in question, and would probably manifest itself more within the boundaries of the comparatively adjacent upper part of the capsule, or of the tracts running from this to the cortex, than within more distant parts; at any rate, it seems that this might occur more readily than that the whole cortical centre for the arm should be affected through remote action. A protracted "remote effect" is, however, improbable in the case of a vascular lesion, whereas a scar or contraction of tissue seems more probable.

In Case XXIII. (narrated in full on p. 467) things, were somewhat different. We have to deal here with a brachial monoplegia well marked proximally; the hand and the leg were apparently only indirectly affected, but in the later stage an increase of the knee-jerk and wrist-jerk was observed. But the scapulo-periosteal reflex was absent. This reflex, it is true, is not constant, and no study has yet been made as to its occurrence under pathological conditions; but it was distinct on the non-paretic side, and a conspicuous difference thus existed between the two sides. I think myself justified in placing this case with the preceding ones when trying to find an explanation. One thing that must be emphasized in this connexion is the difference between the purely cortical and the medullary branches from the arteries of the cortex. A higher pressure is required to fill the medullary branches than the purely cortical ones; an ischæmia consequently asserts itself sooner within the area of supply of the former (Monakow [141]). Case XXIII. was produced by a vascular lesion, probably a thrombosis. Hence it is highly probable that the ischæmia of the centre in which motility was most affected—viz., that of the shoulder—destroyed both cortex and sub-cortex in adjacent parts; on the other hand, the ischæmia asserted itself to a lesser degree, and earliest not in the cortex, but in those parts where it can be most readily established—i.e., the sub-cortex. In this way a cortico-subcortical lesion was brought about in the centre for the shoulder, but a slight, more purely subcortical affection of the adjacent parts; and in this fact might perhaps be found an explanation of the increased reflexes of the forearm and the leg. It would therefore not be necessary to have recourse to remote influence as an explanation. It has not been my intention to set out the above consideration as a definite explanation with regard to these cases, which have only been clinically examined; moreover, the number of cases is much too small for a generalization; still, the suggestion I have made seems to me a possible explanation, especially with the support afforded by Case V., which came to post-mortem examination.

With regard to Monakow's view that the hypertonus becomes less marked when sensory disturbances are present, I wish to point out that this is not evident in my cases. Nor could I establish any connexion between the disorders of sensation and the behaviour of the reflexes. Case XIX., with slight sensory impairment, showed slight increase of reflexes; Case XV., with sensory-motor paralysis, showed normal reflexes; Case XIII., with sensory-motor paralysis, showed slight increase of reflexes; in Case XI. the reflexes were equal on both sides with slight motor paresis; Case X. a purely motor paralysis with moderately increased reflexes; Cases XVI. and IX. evidently increased reflexes were associated in the former case with sensory-motor, in the latter with purely motor, paralysis.

Neglecting those of my cases I have adversely criticized, one point stands out in all the remainder where the expected increase of reflexes did not occur. In every such case the lesion was localized in the cortex.

§ 3.—*The Late Contracture.*

If, then, my cortical cases presented material differences from the capsular ones with regard to the reflexes, this is equally true with reference to contracture — i.e., the typical late contracture. With regard to the so-called "Frühcontractur" the conditions are different, and to this I shall return later.

The late contracture, like the muscular rigidity, must be regarded as a higher degree of the same process that gives rise to the exaggerated reflexes. Hence it is evident that processes that prevent the appearance of reflex hyperexcitability must also prevent the onset of contracture; this makes it unnecessary for me to enter upon them once more. On the other hand, processes may produce the lower degree of spasticity, the reflex hyperexcitability, but not contracture. Such processes are the so-called senile hemiplegia ("hémiplégie des vieillards," Marie), in which, as Ferrand points out, no contractures occur. Here it should, however, be observed that by this name Ferrand denotes a process of quite a different nature—viz., "état lacunaire." Absence of contracture is the rule also in lesions affecting the optic thalamus (Roussy). But with the exception of Monakow's statement I have not been able to find in the recent literature any direct intimation that matters might be different with regard to cortical lesions.

In two older French clinicians I have, on the other hand, found some statements of the utmost importance for this question—viz., in Rostan (1823), and especially in Durand-Fardel (1843 and 1854). As a matter of fact, the latter states positively that, in the chronic stage of a softening, contractures are far less common than in the acute. And if we look at those cases recorded in his work of which a sufficiently detailed account is given, it becomes apparent that in three of them—viz., in 66, 72, and 96—where contracture was lacking, and in one—viz., Case 63—showing traces of contracture, the lesion was cortical. In ten cases, again—viz., 59, 64, 67, 73, 82, 93, 94, 97, 98, 117—where the lesion either did not affect the cortex or occupied the cortex together with a larger part of the medulla, there was "roideur." Although these cases only form a fractional part of those observed by him, and bearing in mind that cases without contracture are badly represented, they show that cortical lesions are not liable to cause contractures. Rostan's statement that in softening, contractures are not met with more than in every twentieth case, and Durand-Fardel's experience that hæmorrhages are more often accompanied by contractures than softenings, tell in the same direction. Such a difference between softening and hæmorrhage is not, as far as I can find, pointed out in the recent literature. Gowers, Mills [129], and Oppenheim [158] do not mention the matter. Dejerine [48] is of opinion that hæmorrhage and softening behave similarly in the chronic stage, and asserts further that cortical lesions have caused a permanent contracture. As to my own cases, I need not here enter again upon the circumstances that may have had an "inhibitory influence" on the increased reflexes, and hence also on the hypertonic state, in Cases VI., VIII., XIV., XVII., XVIII., XX. and XXI. As the contractures do not, as a rule, come on till after six to twelve weeks, a large portion of my material does not prove anything with regard to this question. This holds good with regard to the Cases VII., X., XII., and XXII., as the time of observation in these cases lasted about 2 weeks, 6 weeks, 6 weeks, and one week respectively after the onset. Cases IX., XI., XIII., XV., XVI., and XIX. were under observation respectively 1, ½ year, 11, 4, 3½, 6 months, 1½ year after the onset of the hemiplegia, and in all these cases "Spätcontractur" was lacking. Case IX. presented, it is true, a plantar contracture of the foot, but, considering the fact that there was no hyperexcitability of the reflexes in the leg, I think we can regard it as a passive contracture from the beginning, the more so as the patient had long been confined to her bed before she obtained rational medical attendance.

Case XXIII., again, in connexion with a cortical lesion showed a monoplegia with "Frühcontractur," which quickly disappeared. Two and a half months later there were signs of a slight contracture of the thigh. After the lapse of four months this had become distinct, and after another five months a certain degree of contracture of the hand had also supervened. Since, however, the lesion within the centres for the hand and the leg may be presumed not to have affected the cortex itself, but only its paths of projection, this case does not go against my other experience.

Since I met with contracture in all cases of capsular lesion, but not in cortical cases, the difference is obvious.

§ 4.—*The Early Contracture.*

If, on the other hand, attention is paid to the so-called "Frühcontractur," the matter is totally different. It is necessary strictly to differentiate, as Monakow does, between the real chronic contracture, which comes on late, and the so-called "Frühcontractur," which might rather be put on a par with the effect of cortical irritation. This may be gathered from Munk's experience gained on monkeys, where it often occurred in connexion with a cerebral wound that healed badly, and from the fact that it often occurs in man in connexion with wounds of the cerebral cortex caused by penetrating fragments of bone (Monakow) or after an incision, as in the case related by Liepmann [113].

That early contractures relatively often occur in connexion with softening of the cortex cerebri is, moreover, evident from Durand-Fardel's and Rostan's investigations; and the former points out that this early contracture found in association with softening is not necessarily, or even usually, followed by a chronic contracture.

In my cases there were traces of such an early contracture in three instances, viz., in Case VII., in Case XX., and in Case XXII. The nature of the cataleptic tension observed in Case XIX. cannot be decided, but it is not impossible that it came on in connexion with the convulsive fit of the patient, and in that case must be looked upon as a "Frühcontractur."

Further, I shall relate a case of cortical lesion showing a rather marked "Frühcontractur," the onset and development of which I have had the opportunity of following more closely.

CASE XXIII.—K. G. H.—With regard to the previous health of the patient, it may be mentioned that one year previously, during an attack of dizziness, he fell down a flight of stairs and hurt his right shoulder. Next day, however, he was well and was able to perform his work (compositor) until November 25, 1908.

During the night of November 25 he observed that the right arm was weak and that he had some difficulty in speaking for about an hour and a half. Consciousness was not affected. He was admitted on November 25.

On admission, a considerable paresis was discovered for all movements of the right arm, and a slight paresis of the right lower part of the face. The motility of the leg could not then be examined thoroughly. The patellar reflexes were brisk on both sides. The tendon and periosteal reflexes of the arms were brisk and equal on both sides. The urine contained albumin, 0·4 per cent., with numerous hyaline and granular casts; blood-pressure raised, 170—180 mm. (Riva-Rocci-Landergren).

On December 2 the paresis was found to be greatest at the shoulder-joint and in the muscles of the shoulder in the scapular region; here the paralysis was complete, less at the elbow-joint, and comparatively less again in the joints of the wrist and fingers. The patient was, however, unable to perform isolated movements of the fingers. On the same occasion an intense active contracture was discovered affecting all the joints of the right arm to movement in all directions, but especially the flexors of the elbow, wrist, and fingers. No paresis of the face or of the leg was present.

Active motion of the hand returned quickly, both for coarse movements (dynamometer on the right and left side respectively December 7, 12 and 18; December 11, 18 and 20—21; December 12, 20 on both sides), and with regard to the power of isolated movements of the fingers (December 2, no power of isolated movements; December 4, isolated movements of the thumb present; December 7, also those of the index- and middle-fingers; and, lastly, on December 10, of all the fingers).

On December 2 there was at the elbow-joint slight power of flexion, but almost none of extension. The power increased fairly equally in both directions of movement, but still at the close of the time of observation the power of the right elbow-joint was less good, especially with regard to extension, than that of the left one.

At first there was a complete paralysis of the movements of the arm; on December 7 there were slight power of adduction and rotation inwards, which on December 10 had considerably increased; a slight degree of motility in other directions was present. During the further course of the disease the power of all the movements of the arm increased somewhat, returning to a relatively greater extent for adduction, inward rotation, and extension, although these movements remained considerably weaker than on the other side; a relatively greater paresis remained in the movements of the arm than in those of the forearm.

The initial contracture diminished more quickly than the paresis. It was, to begin with, distinct in all the muscles of the upper extremity, but much stronger in certain groups—viz., in the flexors of the finger, wrist, and elbow-joints; in the shoulder it was most pronounced in the antagonists to outward rotation, adduction, and flexion. The contracture passed off at about the same time in all the joints, but differently for different directions of movement, asserting itself longest in those directions where it had been most pronounced. On December 7 the contracture had much decreased, but after this remained unaltered until December 14; on December 17 it was slight, and had disappeared on December 27. During a period of about one month thereafter there were no signs of contracture. On February 6 a trace of contracture was, however, noticed in the extensor muscles of the thigh. This contracture, which had developed later, increased during the next three months, but did not attain any higher degree even during this time. Five and a half months after the onset there were slight signs of contracture both of the wrist and fingers, especially of the flexors. It must, however, be remarked that long before the appearance of any rigidity in connexion with passive movements of the wrist and finger-joints the active movements showed a certain degree of sluggishness, which increased as soon as the rigidity had manifested itself in passive movements. While the contracture lasted, considerable pain was produced by movements of the shoulder-joint. The pain was more pronounced when the muscles which were the seat of the greater contracture were being stretched, and it disappeared at the same time as the contracture.

It should be added that the shoulder-joint itself was found on examination to be free from abnormal changes. The tendon reflexes were at first equally brisk on both sides until December 12; on December 14 and 18 the reflexes of the right arm were brisker, and constantly the radio-periosteal reflex was the one that was most increased. The scapulo-periosteal reflex, again, was absent for a long time on the paretic side, but was constant on the non-paretic side. The patellar reflex was somewhat brisk, but equally so on both sides, until, on December 27, when a greater exaggeration could be observed on the right side; at this time the tendo-Achillis reflex was equal on the two sides. This remained when the patient was discharged, and on February 6 a slight contracture of the leg was also noticed; the foot was dragged along the floor at each step, in spite of the fact that there was no paresis in the movement of dorsal flexion of the foot. Babinski's sign was present at the first examination on November 25 in the right foot, but could not afterwards be elicited until February 6. The abdominal reflexes were not obtained. On May 6 the patellar reflex on the right side was much increased, and a hypertonic condition of the muscles of the thigh was evident on the same date. The Achillis-tendon reflex was equal on both sides. No Babinski's sign was obtained, and no hypertonus was discovered in the muscles of the calf.

The examination of sensation in this case was very difficult to make; at any rate, during the earlier period sensibility seemed to be diminished in the right arm, but on December 27 there was no astereognosis or asymbolism.

In this case the patient had a cerebral seizure accompanied by paralysis of the arm. From the history of the patient's illness it appears that motility was quickly restored in the distal parts, but slowly and incompletely proximally. Upon this point, however, I shall not now enter. The cause may with certainty be attributed to a vascular lesion, situated within the cortex within the upper part of the centre for the arm.

In this case passive movements evoked pain in the shoulder-joint. The first question, then, to decide is obviously whether this was due to arthritis which may follow cerebral paralysis; but it does not generally occur acutely. In Darkschewitsch's forty-five cases it appeared from one to six months after the seizure. In addition to the pain evoked by movement, tenderness is generally present, and even a palpable swelling of the joint. In my case the pain came on immediately after the seizure, but the joint itself presented no changes, the pain only appearing in connexion with movements, especially such movements as affected the contracted muscles. This also coincides with Darkschewitch's experience, who, before the appearance of any signs of arthritis, found that pain was only caused when the pectoral muscle, the seat of a contracture, was being stretched. Hence I regard it as highly probable that the pains in my case were due to the contracture, especially as they disappeared simultaneously with it.

The contracture, which was here observed as early as a week after the seizure, was apparently a so-called early contracture in Monakow's sense—a tonic cramp—and therefore a real sign of irritation. It here affected all the muscles of the extremity, but, as is often the case, had affected those muscles most which are more particularly the seat of the common late contracture—that is to say, the adductors and inward rotators of the shoulder-joint and the flexors of the elbow-joint. Further, it appeared that the contracture had nothing to do with the degree of loss of power or its return, but disappeared, on the contrary, almost simultaneously in all the joints. It was not followed by any late contracture of the shoulder-joint, the part most affected from the beginning; whereas the leg, which had not shown any paresis, became the seat of a slight late contracture.

It was first difficult to decide which muscles of the leg were the seat of the late hypertonus, as it did not manifest itself in connexion with passive movements, but only in walking. As, however, a hypertonic condition and a reflex hyperexcitability generally go together, it was regarded as more probable that the hypertonus was situated within the region of the reflex arc of the knee-jerk—i.e., within the muscles of the thigh—than within that of the tendo-Achillis jerk—i.e., within the muscles of the calf; this was also borne out by its subsequent behaviour. In this case there was also an obvious increase of the reflexes, but this did not appear in the arm until more than two weeks after the seizure, and showed itself later still in the knee-jerk and not at all in the tendo-Achillis reflex.

As regards the deep reflexes of the arm, as the distal and the proximal motility were different, so also the increase of the reflexes was constantly greatest in the distal parts. This might perhaps be connected with the fact that the lesion had gravely affected the foci of more proximal parts, whereas, judging from all previous experiences, it cannot have affected the centre for the hand itself. If such had been the case the finer motility of the hand would not have been restored so completely. In the leg the reverse condition prevailed; here the exaggerated reflexes were found only proximally, whereas distally the reflexes were normal. This, it seems to me, might well be explained by the circumstance that the influence of the lesion situated within the centre for the shoulder-joint reached the parts of projection of the proximal portion of the leg, but not the more remote paths of projection belonging to its distal parts.

It is, anyhow, interesting to note that this considerable increase of the radio-periosteal reflex did not appear until the motility of the hand had almost returned to the normal state, and, with regard to the leg, no paresis could be discovered even at the onset of the disease; but, in spite of this, a late increase of the reflexes made its appearance. I have already tried to give an explanation of these facts, to which I here beg to refer (p. 464).

The Babinski reflex was here present at first, but was afterwards absent during the whole period of the early contracture, which, according to Monakow, is a common phenomenon; it did not return until a long time after the contracture, but later than the increase of the patellar reflex.

In this case we find, then, as the result of a cortical lesion, a monoplegia with increase of the reflexes, partly in the monoplegic arm, partly in the leg, which was, however, not paretic; the increase of the reflexes was greatest in that part of the arm the cortical centre for which cannot have been the seat of a destruction, but which has been indirectly affected, like the leg. This increase of the reflexes came on in spite of the absence of paresis.

Further, there was in this case an early contracture of the whole arm, most definite in those muscles usually first affected by late contracture; on the other hand, a slight hypertonus of the leg was observed, which was only secondarily affected. This early contracture disappeared rather quickly, and a slight rigidity of the joint of the wrist and the fingers, but not of the elbow-joint, appeared, although not until much later. Further, a spastic and hypertonic condition of the upper part of the leg developed about two and a half months after the attack, although the part did not appear to be in any way affected after the initial seizure.

This case shows clearly, as Monakow has pointed out, that early contracture, in his sense, and late contracture are two different things, and that an early contracture may very well develop in connexion with a cortical vascular lesion.

In association with cortical vascular lesions it seems, then, to be a common phenomenon that the complex of spastic symptoms which otherwise accompanies uncomplicated supranuclear lesions is materially modified, or even fails to develop. This holds good, firstly with regard to the secondary active contracture which failed to appear in all my cortical cases. In three cases, on the other hand, more or less marked early contracture was noticed. But the modification of the spastic symptoms also affects the reflexes, which in two cases only out of twelve showed an increase, comparable to that usually found in typical hemiplegia. In both this increase occurred in that extremity which was not directly affected by the paralysis, whereas four cases presented only a slight increase of the reflexes, four cases equal reflexes on both sides, and two cases even a diminution of the reflexes on the paretic side.

That the cortical localization of a lesion, irrespective of its nature, does not always give rise to a similar modification of the spastic symptoms, is best shown by Spilmeyer's case, an intracortical hemiplegia with typical contractures in a person the subject of epilepsy, where nevertheless the pyramidal tract was intact; and that this modification of the spastic symptoms is not without exceptions even in the case of vascular lesions involving the cortex is shown by Dejerine's case, an extensive cortical lesion causing total spastic hemiplegia with contracture.

In conclusion, I believe, however, that my cases here recorded show that in cerebral monoplegia, following a vascular lesion, the spastic signs may be slightly marked only or altogether absent without it being possible to find any other cause than the situation of the lesion in the cortex. Should spastic signs appear at all, they appear much later than in ordinary cases of hemiplegia due to lesions situated elsewhere than in the cortex.

CONCLUSIONS.

(1) Motion and sensation are not localized in the same cortical centres

(2) The cortical centre for motion is situated entirely in front of the sulcus centralis and is limited to the anterior central gyrus.

(3) Sensation is localized entirely behind the sulcus centralis and is situated in the gyrus centralis posterior, and a part at least of the parietal lobe.

(4) The sensory projection-area is subdivided into centres for the various parts of the body, as we know to be the case within the motor area. But it is an open question whether there are separate centres for the various sensory qualities, such as the cutaneous and muscular senses

(5) Cortical lesions do not produce definite increase of the reflexes or the typical late contracture found in hemiplegia of capsular origin. On the other hand, early contracture ("Frühcontractur") is a common result of cortical lesions and is due to cortical irritation.

LITERATURE.

[1] Aba. "Etude clinique des troubles de la sensibilité générale des sens musculaire et stéréognostique dans les hémiplégies de cause cérébrale," Thèse, Paris, 1896.
[2] Albertoni and Brigatti. "Rivista sperimentale di Fren." 1893, *cit.* Dana.
[3] Alessandri. *Monatschr. f. Psych. und Neur.*, 1906, vol. xviii, p. 62.
[4] Alrutz. "Allmänna svenska läkaretidningen," 1907, vol. iv, p. 841.
[5] Barker. *New York Med. Journ.*, 1892, *cit.* Dana.
[6] Bartholow. *Amer. Journ. Med. Sci.*, 1874, *cit.* Brécy.
[7] Bastian. *Brain*, 1888, vol. x, p. 1.
[8] Beevor. *Brain*, 1907, vol. xxx, p. 407.
[9] Beger. *Archiv f. Heilk.*, vol. xix, *cit.* Nothnagel.
[10] Benedict. *Wien. klin. Wochenschr.*, 1907, vol. xx, p. 66.
[11] Bickeles. *Neur. Centralbl.*, 1901, vol. xx, p. 296.
[12] Binswanger. "Charité Annalen," 1883, vol. viii, p. 498.
[13] Blix. "Upsala Lakareförenings förhandlingar," 1884, vol. xix, p. 123.
[14] Bonhoeffer. *Monatschr f. Psych u. Neurol.*, 1898, vol. iii, p. 297.
[15] *Idem.* *Deutsche Zeitschr. f. Nervenheilk.*, 1904, vol. xxvi, p. 57.
[16] Bourdicaud-Dumay. "Recherches cliniques sur les troubles de la sensibilité générale du sens musculaire et du sens stéréognostique dans les hémiplégies de cause cérébrale." Thèse, Paris, 1897.
[17] Bramwell. *Brain*, 1899, vol. xxii, p. 1.
[18] *Idem.* *Edin. Med. Journ.*, 1878, p. 141, *cit.* Lisso.
[19] Brécy. "Les troubles de la sensibilité dans l'hémiplégie d'origine cérébrale," Thèse, Paris, 1902.
[20] Bremer and Carson. *Amer. Journ. of Med. Sci.*, 1892, *cit.* Dana.
[21] Brieger. *Berl. med. Wochenschr.*, 1887, vol. xxiv, p. 882.
[22] Broadbent. *Lancet*, 1878, vol. i, p. 312.
[23] Brodmann. *Journ. f. Psych. u. Neurol.*, 1904-5, vol. iv, p. 177.
[24] *Idem.* *Journ. f. Psych. u. Neurol.*, September, 1907.
[25] Bruns. *Neurol. Centralbl.*, 1898, vol. xvii, p. 770.
[26] *Idem.* "Die Geschwülste des Nervensystems," 1908.
[27] Brugsch and Schittenhelm. "Klinische Untersuchungsmethoden," 1908.
[28] Bullard. *Journ. of Nerv. and Ment. Dis.*, 1904, vol. xxxi, p. 241.
[29] Burr. *Amer. Journ. Med. Sci.*, 1901, vol. cxxi, p. 304.
[30] *Idem.* *New York Med. Journ.*, 1903, vol. lxxviii, p. 689.
[31] Bychowski. "Congrès international de Psychiatrie," &c., Amsterdam, 1907, pp. 462 and 492.
[32] Cadwalader. *Journ. of Nerv. and Ment. Dis.*, 1908, vol. xxxv, p. 329.
[33] Cajal. "Studien über die Hirnrinde des Menschen," 2 Heft; "Die Bewegungsrinde," Leipzig, 1900.
[34] *Idem.* *Ibid.*, 5 Heft., "Vergleichende Strukturbeschreibung," &c., Leipzig, 1906.
[35] Campbell. "Histological Studies on the Localization of Cerebral Function," Cambridge, 1905.
[36] Carson. *Courier of Med.*, 1891, *cit.* Dana.
[37] Charcot. "Leçons de Mardi, 1887-88," Paris, 2nd edit., p. 209.
[38] *Idem.* "Maladies du système nerveux," iii., Paris, 1887.
[39] Collier and Batten. *Brain*, 1899, vol. xxii, p. 473.
[40] Collier. *Brain*, 1904, vol. xxvii, p. 590.
[41] Cornil. *Gaz. méd.*, 1864, *cit.* Lisso.
[42] Dana. *New York Med. Rec.*, 1893, vol. xliii, p. 578.
[43] *Idem.* *Journ. of Nerv. and Ment. Dis.* (*a*) 1888, vol. xv, p. 650, and (*b*) 1894, vol. xxi, p. 761.
[44] Darkschewitsch. *Neurol. Centralbl.*, 1890, vol. ix, p. 714.
[45] *Idem.* *Arch. f. Psych.*, 1892, vol. xxiv, p. 534.

[46] Dejerine, Sollier and Auscher. *Arch. de Physiol.*, 1890.
[47] *Idem.* "Anatomie des centres nerveux," i, ii, Paris, 1893, 1901.
[48] *Idem.* "Semiologie de système nerveux" in "Traité de Pathologie générale" (Bouchard), 1901, vol. v, p. 359.
[49] *Idem.* *Revue neurol.*, 1893, p. 51, *cit.* Verger.
[50] *Idem.* *Soc. de biol.*, 6/4, 1895, *cit.* Long.
[51] *Idem.* *Rev. neurol.*, July 15, 1906.
[53] Dercum and Keen. *Journ. of Nerv. and Ment. Dis.*, 1903, vol. xxx, p. 747.
[54] Diller. *Brain*, 1901, vol. xxiv, p. 649.
[55] Durand-Fardel. "Traité du ramollissement du cerveau," Paris, 1843.
[56] *Idem.* "Maladies des vieillards," Paris, 1854.
[57] Durante. *Brit. Med. Journ.*, 1902, vol. ii, p. 1822
[58] Edinger. *Deutsche Zeitschr. f. Nervenheilk.*, 1898, vol. i, p. 262.
[59] *Idem.* *Arch. f. Psych. u. Nervenk.* 1879, vol. x, p. 83, *cit.* Lisso.
[60] Egger. *Rev. neurol.*, 1908, vol. xvi, p. 345.
[61] Exner. "Functionen in der Grosshirnrinde," Wien, 1881.
[63] Fischer. *Monatschr. f. Psych. u. Neur.*, 1906, vol. xviii, p. 97.
[64] Flechsig. *Neurol. Centralbl.*, 1898, vol. xvii, p. 977.
[65] *Idem.* *Neurol. Centralbl.*, 1903, vol. xxii., p. 202.
[66] *Idem* and Hosel. *Neurol. Centralbl.*, 1890, vol. ix., p. 417.
[67] Förster. *Monatschr. f. Psych. u. Neurol*, 1901, vol. ix., pp. 31, 131.
[68] *Idem.* *Berl. klin. Wochenschr.*, 1909.
[69] Frank and Church. *Amer. Journ. Med. Sci.*, 1890, *cit.* Dana.
[70] v. Friedlander and Schlesinger. *Mitteil. aus den Grenzgebieten*, 1898, vol. vi., p. 297.
[71] Ganault. "Contribution à l'étude de quelques réflexes dans l'hémiplégie d'origine organique," Thèse, Paris, 1898.
[72] Goldflam, *cit.* Oppenheim.
[73] Gordon. *Journ. of Nerv and Ment. Dis.*, 1908, vol. xxxv, p. 329.
[74] *Idem.* *Ibid.*, 1903, vol. xxx, p. 144.
[75] Gowers. "Diseases of the Nervous System," vol. ii, London, 1893.
[76] Graeffner. *Münch. med. Wochenschr.*, 1906, vol. liii, p. 489.
[77] Grasset. *Rev. de méd.*, 1880, *cit.* Redlich.
[78] Gray. *Brain*, 1892, vol. xv, p. 443.
[79] Grisson and Sanger. *Neurol. Centralbl.*, 1898, vol. xvii, p. 1138.
[81] Guillain. Ref. in *Rev. neurol.*, 1907.
[82] Göthlin. "Om ledningens natur i hvita substansen," Thèse, Upsala, 1907.
[83] Hammarberg. "Studier ofver Idiotiens Klinik och Patologi," Thèse, Upsala, 1893.
[84] Hatschek. *Jahrbucher f. Psych. u. Neurol*, 1905, vol. xxvi, p. 253.
[85] Henschen. "Klinische und anatomische Beitrage zur Pathologie des Gehirns," i, ii, iii, iv, 1890-1903.
[86] Herbold. *Deutsch. med. Wochenschr.*, 1894, vol. xx, p. 511.
[87] Holmes and Page-May. *Proc. Roy. Soc. Med.*, 1909, vol. ii, part 5 (Neurol. Section), p. 92.
[88] Hoppe. *Journ. of Nerv. and Ment. Dis.*, 1904, vol. xxxii, p. 281.
[89] Horsley, *cit.* Marinesco, *Sem. méd.*, 1896.
[90] *Idem.* *Brit. Med. Journ.*, 1886, vol. ii, p. 670.
[91] *Idem* and Schafer. *Philosoph. Trans.*, 1888, p. 179.
[92] *Idem* and Beevor. *Philosoph. Trans.*, 1890, pp. 49 and 129.
[93] *Idem* and Russel. *Brain*, 1906, vol. xxix, p. 137.
[94] *Idem* and Slinger. *Brain*, 1906, vol. xxix, p. 1.
[95] Hudson. "Annals of Surgery," 1893, *cit.* Dana.
[96] Hösel. *Arch. f. Psych. u. Nervenk.*, 1892, vol. xxiv, p. 452.
[97] *Idem.* *Arch. f. Psych. u. Nervenk.*, 1905, vol. xxxix, p. 195.
[98] Infeld. *Wien. klin. Wochenschr*, September, 1900, No. 44.
[99] Jany. *Arch. f. Augenheilk.*, 1882, vol. xi, p. 190.

[100] KAHLER and PICK. *Prager Vierteljahrsschrift*, 1879, *cit.* NOTHNAGEL.
[101] A. KNAPP. *Monatschr. f. Psych. u. Neurol.*, 1908, vol. xxiii, Erg. heft, p. 16.
[102] C. KNAPP. "Contribution to Cerebral Localization," 1891, *cit.* SOURY.
[103] KRAMER. *Monatschr. f. Psych. u. Neurol.*, 1906, vol. xix, p. 129.
[104] KRAUSE. "Hirnchirurgie," *Die deutsche Klinik*, 1905, vol. viii., p. 953.
[105] KRONLEIN. *Correspondenzbl. f. schweiz. Aertz.* 1882, vol. xii, *cit.* v. MONAKOW.
[106] *Idem.* *Arch. f. klinische Chirurgie*, 1901, vol. lxiv, p. 108.
[107] LAYCOCK. *Brain*, 1893, vol. xiv, p. 605.
[108] LENNMALM. "Upsala Lákareforeningens forhandlingar," 1886-87, vol. xxii, p. 299.
[109] LÉPINE. *Progrès Méd.*, 1877, *cit.* REDLICH.
[110] *Idem.* *Rev. de med.*, 1883, *cit.* FISCHER.
[111] LEVI. *Neurol. Centralbl.*, 1903, vol. xxii, p. 947.
[112] LEYDEN. *Deutsch. med. Wochenschr.*, 1837, vol xiii, p. 1013.
[113] LIEPMANN. *Monatschr. f. Psych. u. Neurol.*, 1898, vol. iii, p. 407.
[114] *Idem.* *Monatschr. f. Psych. u. Neurol.*, 1905, vol. xvii, p. 289, and 1906, vol. xix, p 147.
[115] *Idem.* "Drei Aufsätze aus dem Apraxiegebiet," Berlin, 1908.
[116] *Idem.* *Neurol. Centralbl.*, 1904, vol. xxiii, p. 686.
[117] LISSO. "Lokalisation des Gefuhls in der Gehirnrinde," Inaug. diss., Berlin, 1882.
[118] LONG. "Les voies centrales de la sensibilité générale," Thèse, Paris, 1899.
[119] *Idem.* "Nouvelle Iconographie de la Salpêtrière," 1907, vol. xxi, p. 37.
[120] *Idem* and NOÏCA. *Rev. Neurol.*, 1908, vol. xvi, p. 1332.
[121] LOWY. *Monatschr. f. Psych. u. Neurol.*, 1905, vol. xviii, Erg. heft, p. 372.
[122] MADDEN. *Journ. of Nerv. and Ment. Dis.*, 1893, vol. xx, p. 125.
[123] MANN. "Congrès de Psychiatrie," &c., Amsterdam, 1907, p. 423.
[123a] *Idem.* *Monatschr. f. Psych. u. Neurolog*, 1898, vol. iii.
[124] MARBURG. *Jahrbücher f. Psych. u. Neurol.*, 1905 (Sept.), vol. xxvi.
[125] MARIE. *Rev. de méd.* 10/4, 1901, *cit.* MARIE, "Exposé des titres et travaux scientifiques."
[126] *Idem* and GUILLAIN. *Sem. med*, 1902, vol. xxii, p. 209.
[127] MARINESCO. *Sem. med.*, 1903, vol. xxiii, p. 325.
[128] MARKOWA. "Contribution à l'étude de la perception stéréognostique," Thèse, Genève, 1900, *cit.* *Jahresb. f. Neurol. u Psych.*
[129] MILLS. "The Nervous System and its Diseases," 1897.
[130] MILLS and PFAHLER. *Neurol. Centralbl.*, 1903, vol. xxii, p. 175.
[131] *Idem.* "The Focal Diagnosis of Operable Tumours of the Cerebrum," "Tumours of the Cerebrum," No. 1, Philadelphia, 1906.
[132] *Idem*, KEEN and SPILLER. *Journ. of Nerv. and Ment. Dis.*, 1900, vol. xxvii, p. 224.
[133] *Idem*, FRAZIER. *Journ. of Nerv. and Ment. Dis.*, 1908, vol. xxxv, p. 481.
[134] *Idem.* "The Motor Area of the Human Cerebrum," "Tumours of the Cerebrum," No. 7, Philadelphia, 1906.
[135] MILLS. *Amer. Med. Assoc.*, 1902, vol. xxxix, p. 828.
[136] *Idem* and SPILLER. *Journ. of Nerv. and Ment. Dis.*, 1907, vol. xxxiv, pp. 560, 624.
[137] *Idem* and WEISENBURG. *Journ. of Nerv. and Ment. Dis.*, October, 1906.
[138] MOCUTCHOWSKY. "Nouvelle Iconographie de la Salpêtrière," 1898, vol. xi, p. 230.
[139] v. MONAKOW. *Arch. f. Psych. u. Nervenk.*, 1895, vol. xxviii, pp. 1, 386.
[140] *Idem.* "Ergebnisse der Physiologie," 1902, 1, 2, p. 534.
[141] *Idem.* "Gehirnpathologie," II Auflage, 1905.
[142] *Idem.* *Arch. f. Psych.*, Bd. xxiii.
[143] MORAX. *Bulletins de la Société anatomique de Paris*, 1891, Sér. 5, tome 5, p. 262.
[144] MYNTER. "Annals of Surgery," 1894, *cit.* DANA.
[145] MULLER. Über Storungen der Sensibilitat bei Erkrankungen des Gehirns," *Samml. klin. Vortr.*, No. 394 95, 1905.
[146] MURATOW. *Neurol. Centralbl.*, 1898, vol. xvii, p. 59.
[147] MUSKENS. *Arch. f. Psych. u. Nervenk.*, 1903, vol. xxxvi, p. 347.

[148] Nagel. "Die Lage-, Bewegungs-, und Widerstandsempfindungen," Nagel's Handbuch der Physiologie des Menschen, 1905, vol. iii, p. 735.
[149] Nason, *cit.* Dana.
[150] Noïca. *Rev. Neurol.* 1908, vol. xvi, p. 326.
[151] Nothnagel. "Topische Diagnostik der Gehirnkrankheiten," Berlin, 1905.
[152] *Idem.* "Verhandlungen des VI Congr. f. innere Medizin," Weisbaden, 1887, p 109
[153] Öhrvall. "Upsala Läkareförenings forhandlingar, ny följd," vol. v, p. 1.
[154] Oppenheim. *Charité-annalen*, 1885, vol. x, p. 335.
[155] *Idem.* *Mitteil. aus den Grenzgebieten*, 1900, vol. vi, p. 363.
[156] *Idem.* *Monatschr. f. Psych. u. Neurol.*, 1906, vol. xviii, p. 135.
[157] *Idem.* "Beitrage zur Diagnostik und Therapie der Geschwülste im Bereich des zentralen Nervensystems," Berlin, 1907.
[158] *Idem.* "Lehrbuch der Nervenkrankheiten," 5th edition, Berlin, 1908.
[159] Petrén. *Deutsche Zeitschr. f. Nervenheilk.*, 1900.
[160] *Idem* and Carlstrom. *Deutsche Zeitschr. f. Nervenheilk.*, 1904, vol. xxvii, p. 465.
[161] *Idem* and Bergmark. *Zeitschr. f. klin. Med.*, vol. lxiv, p. 1907.
[162] Petrina. *Zeitschr. f. Heilk.*, 1881, vol. ii, p. 375.
[163] Pfaundler. "Handbuch der Kinderheilkunde," Bd. i, p. 162.
[164] Pierret. *Lyon méd.*, January 31, 1886, *cit.* Verger.
[165] Prince. *Journ. of Nerv. and Ment. Dis.*, 1908, vol. xxxv, p. 1.
[166] *Idem.* *Journ. of Nerv. and Ment. Dis.*, 1908, vol. xxxv, p. 12.
[167] Prohazka, *cit.* Oppenheim.
[168] Ransom. *Brain*, 1892, vol. v, p. 437.
[169] Redlich. *Wien. klin. Wochenschr.*, 1893, vol. vi.
[170] *Idem.* *Ref.* in *Neurol. Centralbl.*, 1908.
[171] Redtenbacher. *Aerztl. Ber. d. k.k. Allg. Krankenh. zu Wien*, 1889-91, p. 66.
[172] Reh. *Monatschr. f. Psych. u. Neurol.*, 1904, vol. xiv, p. 182.
[173] Renvers. *Berl. klin. Wochenschr.*, 1888, vol. xxv, p. 306.
[174] Reymond. *Bull. de la Soc. Anat. de Paris*, Sér. 5, vol. vi, p. 3.
[175] Riegner. *Deutsche med. Wochensch.*, 1894, vol. xx, p. 497.
[176] Rossi and Roussy. *Rev. neurol.*, 1907, vol. xv, p. 785.
[177] Rostan. "Recherches sur le Ramollissement du Cerveau," Paris, 1823.
[178] Rothmann. *Arch. f. Anat. u. Physiol.* (Physiologische Abtheilung), 1907 (Sept.), p. 217.
[179] Roussy. "La couche optique," Paris, 1907.
[180] Roy and Peterson. *Southern Med. Rec.*, 1890, *cit.* Dana.
[181] Sandberg. *Deutsche Zeitschr. f. Nervenheilk.*, 1905, vol. xxx, p. 149.
[182] Savill. *Brain*, 1891. vol. xiv, p. 270.
[183] Schaffer. *Archiv f. Psych. u. Nervenkrank.*, 1908, vol. xliv, p. 228.
[184] *Idem.* *Neurol. Centralbl.*, 1905, vol. xxiv, p. 591.
[185] Schittenhelm. *Deutsch. Arch. f. klin. Med.*, 1906, vol. lxxxv, p. 462.
[186] Sherrington. "The Integrative Action of the Nervous System," London, 1907.
[187] Sherrington and Grunbaum. *Roy. Soc. Proc.*, 1901, vol. lxix, p. 206.
[188] Souques. "Nouvelle Iconografie de la Salpêtrière," 1907, vol. xx, p. 365.
[189] Soury. "Système nerveux central," Paris, 1900.
[190] Spiller. *Journ. of Nerv. and Ment. Dis.*, 1900, vol. xxvii, p. 156.
[191] *Idem.* *Amer. Journ. of Med. Sci.*, 1904, vol. cxxvii, p. 293.
[192] *Idem.* *Journ. of Nerv. and Ment. Dis.*, 1899, vol. xxvi, p. 43.
[193] *Idem.* *Journ. of Nerv. and Ment. Dis.*, 1906, vol. xxxiii, p. 117.
[194] Spilmeyer. *Münch. med. Wochenschr.*, 1906, vol. liii, p. 1404.
[195] Starr and McCosh. *Amer. Journ. of Med. Sci.*, 1894, vol. civ, p. 517.
[196] *Idem* and McBurney. *Brain*, 1891, vol. xiv, p. 284.
[197] *Idem.* "Brain Surgery," p. 38, Case 5, *cit.* Dana.
[198] Stauffenberg. *Arch. f. Psych.*, 1909, vol. xlviii, p. 683.
[199] Steele. *Journ. Amer. Med. Assoc.*, 1894, *cit.* Dana.
[200] Steinert. *Deutsch. Arch. f. klin. Med.*, 1906, vol. lxxxv, p. 445.

[201] STERNBERG. "Die Sehnenreflexe," Leipzig and Wien, 1893.

[202] STEWART GRAINGER. *Brit. Med. Jour.*, 1894, vol. i, p. 1.

[203] STEWART PURVES. *Rev. of Neurol. and Psych.*, 1908, vol. vi, p. 379.

[204] STRAUSSLER. *Monatschr. f. Psych. u. Neurol.*, 1908, vol. xxiii, p. 381.

[205] STRUMPELL. "Spezielle Pathologie und Therapie," 16th edition, Leipzig, 1907.

[206] *Idem.* *Deutsche med. Wochenschr.*, 1904, vol. xxx, pp. 1411 and 1460.

[207] SÖDERBERGH. *Hygiea*, 1908, vol. lxxix, p. 616.

[208] TELLER and DERCUM. *Journ. of Nerv. and Ment. Dis.*, 1901, vol. xxviii, p. 459.

[209] THOMAS. *Brit. Med. Journ.*, 1894, *cit.* v. MONAKOW.

[210] THUNBERG. "Physiologie der Druck-, Temperatur- u. Schmerzempfindungen," in Nagel's Handbuch der Physiologie des Menschen, 1905, vol. iii, p. 647.

[211] *Idem.* *Deutsche Zeitschr. f. Nervenheilk.*, 1904, vol. xxviii, p. 59.

[212] TIGERSTEDT. "Lehrbuch der Physiologie des Menschen," 2nd Edition, vol. ii, Leipzig, 1902.

[213] TSCHERMAK. "Die Physiologie des Gehirns," in Nagel's Handbuch der Physiologie des Menschen, 1905, Bd. iv, p. 1.

[214] VALKENBURG. *Neurol. Centralb.*, 1906, vol. xxv, p. 594.

[215] VALKENBURG. *Arch. f. Psych. u. Nervenkrank.*, 1907, vol. xliii, p. 899.

[216] WALTON and PAUL. *Brain*, 1901, vol. xxiv, p. 430.

[217] *Idem.* *Journ. of Nerv. and Ment. Dis.*, 1901, vol. xxviii, p. 191.

[218] VERGER. "Des anæsthésies consécutives aux lésions de la zone motrice," Thèse, Bordeaux, 1897.

[219] *Idem.* *Arch. gén. de méd.*, 1900, vol. clxxxvi, p. 675.

[220] *Idem*, and DE BOUCAUD. *Arch. Clin. de Bord.*, 1898, *cit.* VERGER [219].

[221] WERNICKE. "Arbeiten aus der Psychiatrischen Klinik in Breslau," 1895, Heft 2, p. 33.

[222] *Idem* and HAHN. *Virchow's Archiv*, 1882, vol. lxxxvii, p. 335.

[223] WESTPHAL, *Charité Annalen*, 1882, vol. vii, p, 466.

[224] VETTER. *Deutsch. Arch. f. klin. Med.*, 1878, vol. xxii, p. 421, *cit.* LISSO and NOTHNAGEL.

[225] *Idem.* *Ibid.*, 1894, vol. lii, p. 352.

[226] WICHURA. Inaugural dissertation, 1881, *cit.* LISSO.

[227] WILLIAMS. *New York Med. Journ.*, 1892, *cit.* DANA.

[228] WILLIAMSON. *Glasg. Med. Journ.*, 1899, vol. lii, p. 335.

[229] WILSON. *Brain*, 1908, vol. xxxi, p. 164.

[230] *Idem.* *Lancet*, 2892, *cit.* DANA.

[231] VOGT. "Neurobiologische Arbeiten," Jena, 1901, Ser. 1, vol. ii.

[232] ZENNER. *Journ. of Nerv. and Ment. Dis.*, 1886, vol. xiii, p. 438.

Vergleichende Lokalisationslehre der Grosshirnrinde. Von K. BRODMANN. Mit 150 Abbildungen im Text. S. 324. Leipzig: Barth, 1909.

THOUGH Meynert, Betz, Bevan Lewis, and others recognized many years ago that considerable differences exist between the structure of the cortex of different regions of the forebrain, this subject—despite its great interest to the physiologist, psychologist, and even to the clinician—has not been seriously studied till within the last few years. And of the many authors who have devoted their labour to it, none has been more prominent than Brodmann; the care and accuracy that have characterized his work, and the essentially objective nature of his conclusions, have been widely recognized. His results have been the more valuable as, in the first place, he has approached the subject as a pure anatomist, and has not burdened his conclusions by attempts to correlate structure with function when there is as yet insufficient material to warrant it, and as, in the second place, his work has been developmental and comparative as well as histological.

The volume under review is intended to present the principles of comparative cortical localization, and to show its aims and scope. The author consequently warns his readers that if they hope to find in it the details of the histology of the human cortex, they may be disappointed.

The cortex of every brain can be subdivided into numerous areas, each determined by its special structure, according to the arrangement and the character of the cells and myelinated fibres that it contains, or by special characters visible in neuro-fibrillar preparations. Brodmann's work has been almost exclusively on the cells of the cortex, or its *cyto-architecture.* The first chapter is devoted to the lamination of the cortex. He maintains that its original and primitive structure is six-layered in all mammals, and that the six layers are always present either permanently or during some period of development, and all variations can be traced to modifications of this six-layer type; the only exceptions are a few regions in which the cortex remains rudimentary, as in part of the rhinencephalon. The following are the layers he describes; the corresponding terms adopted by Campbell are printed in brackets for the convenience of English readers who are already familiar with Campbell's work:—

(I) Lamina zonalis (plexiform layer).

(II) Lamina granularis externa (layer of small pyramidal cells).

(III) Lamina pyramidalis (the layer of medium-sized pyramids and the external layer of large pyramids).

(IV) Lamina granularis interna (layer of stellate cells).

(V) Lamina ganglionaris (internal layer of large pyramids).

(VI) Lamina multiformis (layer of spindle-shaped cells).

Though the cortex of different regions presents special characters, it may preserve its six-layer type (homotypical), and the special character of each region then depends only on the variable density of the cells in any one layer or in all, on the size or structure of the cells, or on the relative breadth of any one layer or of the cortex as a whole. Or the six-layer type may be modified by the disappearance of a layer, as of the internal granular layer in the anterior central gyrus of the adult, though it is present here in the human fœtus of the eighth month; or by the hypertrophy and sub-division of a layer, as of the same layer in the calcarine region (heterotypical cortex). The layers that show the least regional variation are those that are most constant throughout the mammalian series, and *vice versa.* The laminæ zonalis and multiformis are the most constant, and they alone may represent the cortex, as in the cornu ammonis; the external and internal granular layers, on the other hand, are the most variable.

The structure of the cortex and its regional modifications are governed by the same laws in all mammals, but each class has special peculiarities in both the whole cortex and its constituent fields. Its breadth, as well as the wealth in cells and the size of its cells, which can be of course compared only in homologous regions, has no relation to the position of the animal in the mammalian series, but the breadth is to some extent parallel to the size of the body; and there does not appear to be any definite law regulating the relative development of the individual layers in different classes, though in the lower mammals the three deeper layers, when measured together, are relatively broader as compared with the three superficial layers than in the higher classes.

By the study of its cell structure the human cerebral cortex can be mapped out into about fifty different fields—*areæ cyto-architectonicæ*—but certain of these which lie adjacent to one another can be grouped together by certain general characteristics into *regiones.* In man

Brodmann distinguishes eleven of these larger division as regio frontalis, regio præcentralis, regio postcentralis, &c. The borders of many of these areas are sharp and definite, but the transition between others is gradual and not sharply definable.

The number, size, distribution, and structural peculiarities of these areas in most of the important mammalian classes are described by Brodmann. He finds that there is a remarkable similarity in the topographical cortical localization in different animals, though, despite a general similarity, great variations may occur between even closely allied species. Certain fields, as the area gigantopyramidalis, area striata, area insularis, are strikingly constant in all animals, but even these vary enormously in size in different species. The precentral area that contains the giant (Betz) cells constitutes only about one-hundredth of the whole cortex in man, while in the lower apes it is about a twentieth or a tenth of the whole. Approximately the same relation exists between the size of the area striata in the two classes. On the other hand, the regio frontalis becomes smaller as we descend from man and simpler in structure—that is, fewer fields can be distinguished in it; in man it includes about three-quarters of the frontal lobe, in the lower monkeys its superficial extent only equals that of the regio præcentralis, while in the lower mammals and marsupials it is absent. This progressive differentation—that is, the relative increase in the size of a region and in the number of its fields—occurs in other regions of the brain; the regio postcentralis contains only a single field in the lowest mammals, but three or four separate fields in the primates. But regressive changes also occur. The area striata of the calcarine region is absent in the mole and poorly developed in the rodents, while the regio olfactoria is much less developed in the higher than in the lower mammals. These variations may be evidently attributed to functional adaptation to environment.

The first two sections of the book are chiefly analytical and descriptive; the third deals with the principles of morphological differentiation and its relation to physiology and histo-pathology. The view that is developed here is that as each cortical field is a *tissue-complex*, with a characteristic structure distinct from that of other parts, each area must be regarded as a separate organ, and that therefore the mammalian cortex is an *organ-complex*. Difference in structure suggests a difference in function, and it may be therefore concluded that each morphological area has some special function. Enticing though this hypothesis may be, Brodmann insists that as yet there is no proof that the cortical localization of any function coincides with that of any structural area; he admits, however, that the area gigantopyramidalis—that portion of the precentral cortex that contains Betz cells—corresponds closely with the excitable region.

The older views, as those held by Meynert, Exner, and Wundt, that all cortical cells are similar or identical, and that difference in function depends only on differences in connexion and association, must be discarded; for not only are there many morphological varieties of cells, but cells identical in form and structure are found in homologous regions in different animals, and the evidence of progressive histogenetic differentiation of some cells is beyond doubt.

Similarly, Brodmann insists that the attempts that have been made to correlate a special layer of the cortex with some function cannot be accepted, and that the theories that localize the higher psychical functions to any area are ridiculous, as these are composite processes which can be, at the most, only centred in a group of areas.

This short review may serve to introduce Brodmann's book to those interested in either the theoretical or practical aspects of the structure and functions of the brain. As the author writes in his preface, those who seek in it the details that are necessary for practical anatomical or pathological work on the cerebral cortex may be disappointed, but that the book is not burdened by such details will make it additionally welcome to those who wish to become conversant with the principles and the main facts of the youngest branch of cerebral anatomy. It can be therefore recommended as warmly to the clinician as to the physiologist and anatomist. The text is written clearly and concisely, and the numerous illustrations that it contains are excellently chosen and reproduced. The one regret that may be expressed is that Brodmann has been so sparing in his references to Campbell's work, for the English author, whose work was completed and presented to the Royal Society in 1903, may justly claim to be one of the pioneers of cortical localization by histological methods, more especially as his conclusions were attained by the study of the myelinated fibres as well as of the cells of the cortex. Brodmann, it is true, has differentiated many more structural areas than Campbell described, but there is a remarkable similarity in their results on the main issues, and it can be scarcely hoped that the fifty or so areas that Brodmann has mapped out represent finality.

GORDON HOLMES.

PUBLICATIONS RECENTLY RECEIVED.

[*Notes on a book under this heading do not preclude a subsequent review.*]

Die Dementia Præcox und ihre Stellung zum Manisch-depressiven Irresein. Eine Klinische Studie von Dr. Med. M. URSTEIN. S. 372. Berlin and Wien: Urban und Schwarzenberg, 1909.

The author believes that the line of division between dementia præcox and maniacal-depressive insanity has been too strictly drawn. Cases which showed no katatonic symptoms, and might have been expected to run a benign course, ended in dementia. But in most instances some katatonic symptoms can be discovered, although the condition may otherwise closely resemble a circular insanity. This materially alters the diagnosis and prognosis. He emphasizes the fact that during the state of stupor the mind is working actively, and the patient's acts, however impulsive they may appear, have a motive; moreover, the memory remains excellent. The prognosis is more favourable than has been usually supposed; the more the form assumed approximates to maniacal-depressive insanity, the more likely is it that remissions will occur. If the katatonic state forms the main feature of the disease, remissions enabling the patient to return to life and work are unlikely. The delirious form almost always recovers, often for a considerable period. Finally, a late katatonia completes the clinical picture. The author's material consisted of 524 cases which had been observed for ten years or longer. Of these, thirty are given in full in the second half of the book.

Otitic Cerebellar Abscess. By HEINRICH NEUMANN. Translated by RICHARD LAKE, F.R.C.S., Pp. 156. London: Lewis, 1909.

This small book is full of information, but the reader is much hampered by the want of an index. The causes and pathological anatomy of abscess due to ear-disease are first described; then follows an interesting chapter on Symptoms and Signs where use is made of Bárány's methods of examination. The two sections on Diagnosis and Differential Diagnosis are too short to be useful to the neurologist, and the author assumes the methods of examination are familiar to those for whom the book is written. Now, although the most modern methods of neurological examination may be known to the aurist, the methods demanded for complete examination of the ear are certainly not in daily use among neurologists. The second half of the book is taken up with a description of cases. The translation contains many Germanisms.

Maladies de la Moelle Épinière. Par J. DEJERINE et ANDRÉ-THOMAS. Avec 420 figures intercalées dans le texte. Pp. 827. Paris: Baillière, 1909.

This splendid text-book forms the thirty-fourth volume of the "Nouveau traité de Médecine et de Thérapeutique" by Gilbert and Thoinot. It is extremely easy to read and contains excellent figures. An anatomical and a physiological introduction are followed by a series of chapters dealing with separate diseases, many of which are treated with remarkable fulness. Thus 145 pages are devoted to tabes and 55 to syphilis. This work should be in the hands of all who wish to know the views of the French school on diseases of the spinal cord. Unfortunately it has no index.

Pupillenlehre, Anatomie, Physiologie und Pathologie, Methodik der Untersuchung. Von Professor Dr. LUDWIG BACH. Mit 27 zum Teil farbigen Abbildungen im Text. S. 344. Berlin: Karger, 1908.

A useful gathering together of all that is known about the pupil, with a full bibliography, containing 1778 papers, and a good index. It will form a useful book of reference.

Les Névroses. Par Dr. PIERRE JANET. Pp. 397. Paris: Flammarion, 1909.

If any neurologist wishes to obtain a clear idea of the views of Dr. Janet in small compass, we can strongly recommend this book, which can be carried in the pocket. The first part treats of neuropathic symptoms, the second of neuropathic states. *Idées fixes*, obsessions, doubts, troubles of speech, choreas and tics, hysterical paralyses and phobias, "crises," the hysterical and psychasthenic states are all dealt with in turn clearly and convincingly.

Modern Problems in Psychiatry. By ERNESTO LUGARO. Translated by DAVID ORR, M.D., and R. G. ROWS, M.D., with a foreword by T. S. CLOUSTON, M.D., LL.D. Pp. 305. Manchester: University Press, 1909.

Professor Lugaro treats his subject under six heads. After a general introduction he discusses the psychological and anatomical problems, pathogenesis, etiology, nosological and practical problems in separate chapters. He bases all his explanations of psychical disease on pathological anatomy and the consequent functional alterations. "Problems in Pathogenesis" is a chapter of unusual interest, and reaches most definitely the author's aim of exciting thought. The translation is well carried out.

Vorlesungen zur Einführung in die Experimentelle Pädagogik und ihre psychologischen Grundlagen. Von ERNST MEUMANN. Bd. 2. Mit 13 Figuren. S. 467. Leipzig: Engelmann, 1907.

The first volume of this extensive work was noticed in BRAIN, 1907. This second volume adds eight more lectures and a complete index. The mental work of the child, mental hygiene of school-work and experiments in reading, writing, arithmetic, and drawing, are all considered fully. The final chapter deals with the future development of experimental pedagogy.

A System of Clinical Medicine. By THOMAS DIXON SAVILL, M.D. Second edition revised by the Author. Pp. 963; with many Illustrations. London: Arnold, 1909.

This well-known text-book is based on the principle of considering the main symptoms of disease rather than the nosological groups which usually form the basis of description in text-books. It is written mainly for practitioners and senior students, and the present edition has been brought thoroughly up to date.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of 'Brain.' If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

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